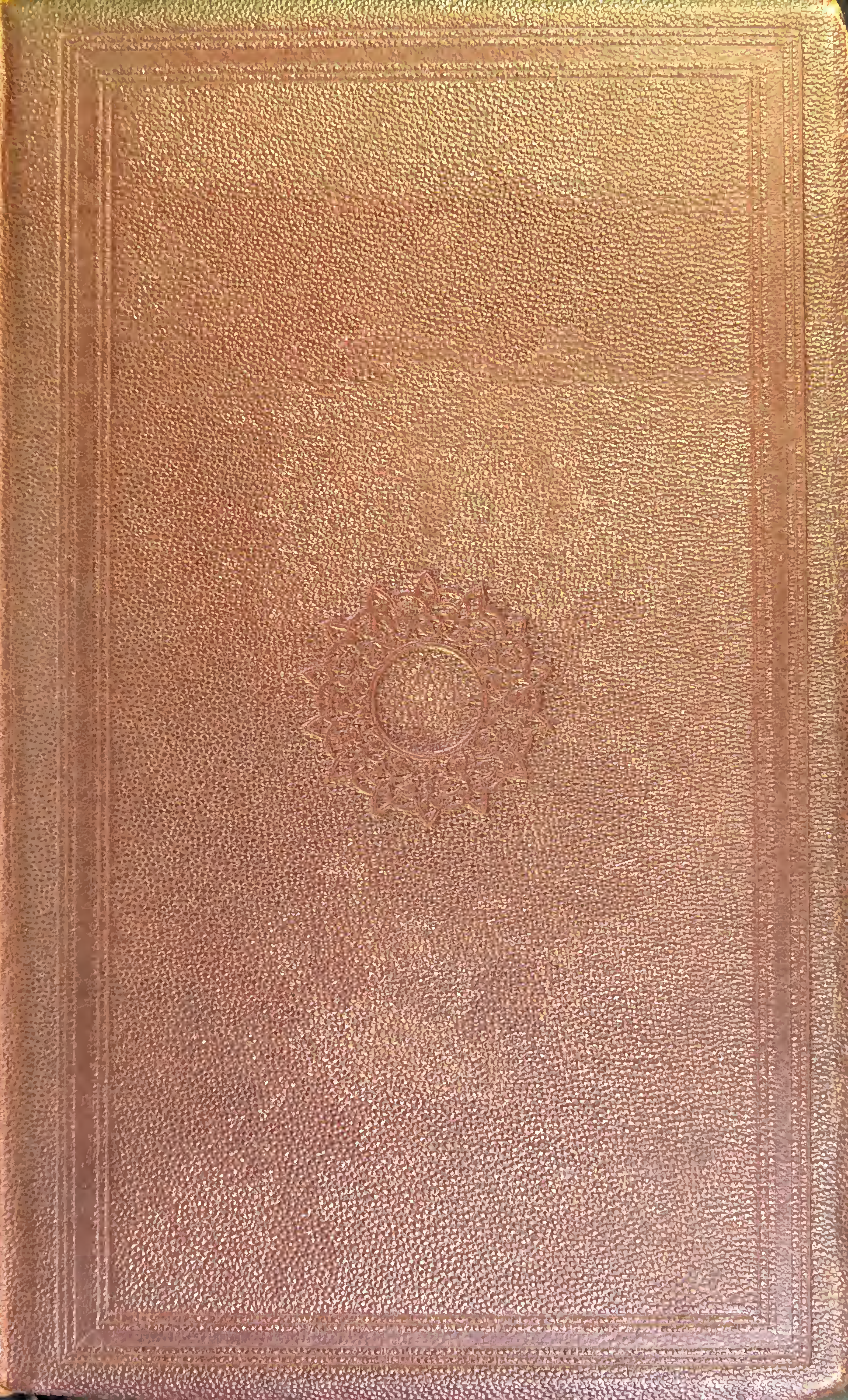

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VOL. XI.

DISEASES OF THE

PERIPHERAL CEREBRO-SPINAL NERVES.

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ERRATA.

VOL. XI.

Page 10, fourteenth line from the bottom, "our" should read "one."

Page 11, thirteenth line from the top, "effects" should read "affects."

Page 28, seventeenth line from the top, the word "by" should be inserted before "all."

Page 34, sixth line from the top, the word "a" should be omitted.

Page 37, last three lines of the page, "them," "Their," and "they" should read respectively "it," "Its," and "it."

Page 60, ninth text-line from the bottom, "easting" should read "lasting."

Page 60, thirteenth text-line from the bottom, "th" should read "the."

Page 66, second and third lines from the top, "spinal cord" should read "vertebral column."

Page 75, fourteenth line from the top, "medicated" should read "mineral."

Page 77, seventh line from the bottom, the word "with" should be introduced before the word "patience."

Page 78, thirteenth line from the bottom, "these" should read "their."

Page 80, eleventh text-line from the bottom, "spiral" should read "coil."

Page 95, ninth text-line from the bottom, "etc." should read "and others."

Page 145, third line from the bottom (in the foot-note), "systematic" should read "schematic."

VOL. IV.

Page 326, thirteenth line from the bottom, "similarly" should read "similar."

Page 600, fourteenth line from the top, "an" should read "any."

Page 723, fifteenth line from the bottom, "An" should read "The."

Page 750, fourteenth line from the bottom, "echinoccus" should read "echinococcus."

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BIOGRAPHICAL SKETCH OF THE AUTHOR.

Wilhelm Heinrich Erb, son of an official forester, was born at Winnweiler, in the Rhenish Palatinate, on the 30th of November, 1840. He attended the gymnasium course of studies at Zweibrücken, and, in the autumn of 1857, entered the University of Heidelberg, where he studied medicine, completing his course in Erlangen and Munich. In 1862 he obtained his degree as Doctor of Medicine in Munich, where, at a later period, he also passed the Bavarian State examination. After serving for six months as Second Assistant to Professor Buhl, at the Pathologico-Anatomical Institute in Munich, he took the position of Assistant Physician at the Clinic of Friedreich in Heidelberg.

In the year 1865 he spent six months in Berlin for the purpose of further perfecting himself in scientific matters, and in the autumn of 1865 was installed as a Private Instructor of Internal Medicine in Heidelberg, at the same time taking the position of first Assistant Physician in the Out-Door Department of Friedreich's Clinic. He delivered lectures on General Pathology, Physical Diagnosis, Human Parasites, etc., and in the year 1868 was nominated Extraordinary Professor. Accidental circumstances led him to make a thorough study of Electrotherapy, for which he had a special interest, and in this way he was naturally led to give particular attention to diseases of the nervous system. During the last few years these two specialties have formed the chief subjects of his studies, and, indeed, of his lectures, since his appointment to lecture on Electrotherapy at the University.

The following are his most important scientific publications:

1. Picric Acid; its Physiological and Therapeutic Actions. Dissert. Wurtzburg, 1864.
2. On the Rise of Bodily Temperature at the Moment of Death in Diseases of the Cerebro-Spinal System. Deustches Archiv f. klin. Med., 1865, Band I., pp. 175–190.
3. Developmental History of the Red Blood-Corpuscles. Habilitation Essay. Virchow's Archiv, Band 34, pp. 138–194. 1865.
4. Occurrence of Trichinæ in Rats. Verh. d. naturhist. Med. Vereins zu Heidelberg, IV., p. 85. 1866.
5. Galvano-Therapeutic Communications. Deutsches Archiv f. klin. Med., Band III. 1867.
6. Case of Facial Paralysis, with Peculiar Irritative Changes in the Paralyzed Muscles. Verh. d. Heidelberger nat. hist. Med. Vereins, IV., 4.

7. On Electrotonic Appearances in Living Persons. Archiv f. klin. Med., Band III. 1867.

8. Remarks on the so-called Waxy Degeneration of Transversely-Striated Muscle-Fibres. Virchow's Archiv, Band 43. 1868.

9. Cases of Nerve and Muscle Disease. Archiv f. klin. Med., Band IV., 1868; V., 1869; VI., 1870.

10. On the Pathology and Pathological Anatomy of Peripheral Paralyses. Archiv f. klin. Med., Band IV., 535–579, and V., pp. 42–95. 1868.

11. The Galvanic Reaction of the Nervous Auditory Apparatus in the Healthy and Diseased Condition. Moos-Knapp's Archiv f. Augen- und Ohrenheilkunde, I. 1869.

12. On the Waxy Degenerations of Transversely-Striated Muscle-Fibres. Archiv f. klin. Med., II. 1869.

13. On the Galvanic Treatment of Eye and Ear Diseases. Moos-Knapp's Archiv, II. 1871.

14. On the Use of Electricity in Internal Medicine. Volkmann's Sammlung klin. Vorträge, 46. 1872.

15. On Tetanus, with Remarks on the Testing of the Irritability of Motor Nerves. Archiv f. Psychiatrie u. Nervenkrankheiten, IV. 1873.

16. On Rheumatic Paralysis of the Facial. Archiv f. klin. Med., XV. 1874.

17. A Case of Lead Poisoning. Archiv f. Psychiatrie und Nervenkrankheiten, V. 1875.

18. On Acute Spinal Paralysis in Adults, and on Allied Spinal Affections. Idem, V. 1875.

19. On the Reflex Twitching of Tendons in Spinal Disease and in Health. Ib., V. 1875.

20. On a Group of Spinal Symptoms that are little Known. Berl. klin. Woch. 1875, 26.

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DISEASES

OF THE

PERIPHERAL CEREBRO-SPINAL NERVES.

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INTRODUCTION.

The great progress that has been made in all departments of neuropathology in the course of the last ten years has not failed to extend materially our knowledge of the diseases of the peripheral nerves. This advance in our information has no doubt been essentially in one direction, that is to say, it has been almost exclusively limited to the accumulation of accurate descriptions of clinical phenomena and to the appropriate application of remedial measures. In comparison to what has been accomplished in this way, the knowledge gained of the pathological and anatomical changes taking place in the peripheral nerves, and of the precise nature of their diseases, has been relatively small, though much has been successfully done. Our insight into the seat and nature of the anatomical lesions in many isolated cases, and even in the most general forms of disease affecting peripheral nerves, is, in fact, still very imperfect, and a glance into the best recent text-books on nervous disorders, in the small space that is given to the anatomical, as compared with that which is devoted to the functional, diseases of this system, indicates clearly enough the state of our present knowledge.

We must still, therefore, preserve the division, so long established, of functional and organic diseases of the nervous system. For we meet, in the first place, with an extensive group of diseases of the peripheral nerves, in which well-marked functional disturbances—as pain, anæsthesia, convulsion, paralysis, and the like—are present, and in which we are unable to point to any constant anatomical lesions; these are consequently termed *functional affections*, or "*neuroses*" of the peripheral nerves; and, in the second place, we have a smaller group, comprising those in which there are distinct anatomical changes, as inflammatory and degenerative processes, and neoplastic formations, which are named "*anatomical*" or "*organic*" diseases of the peripheral nerves.

These two groups present no sharp lines of demarcation, but overlap one another at many points; so that in the first group we meet with certain cases—and, as our information becomes more accurate, such cases occur in constantly increasing number —in which anatomical changes are discoverable, whilst, on the other hand, in cases belonging to the second group, those functional disorders are exhibited which are characteristic of the first group. It is to be hoped that at some future time the two groups will become fused, *i.e.*, that we shall learn to recognize in increasing proportion definite, though perhaps delicate, anatomical changes as constituting the basis of the functional alterations. It must be confessed, however, that this is still a consummation sufficiently distant, however much it may be desired.

In the meantime, whilst admitting the defective state of our knowledge, the necessities of practice compel us to preserve the division hitherto adopted of functional and anatomical diseases. The greater frequency and importance of the former class justifies us in giving to it the first consideration. The account here given will not, however, be limited to functional diseases in the strictest sense of the term, but will include, in consequence of the symptomatological relations which these diseases have with a great number of affections in which there are very marked lesions of the peripheral and central nervous system, many things which ought properly to be included in the neuroses.

Practical considerations and the desire to give a general and complete representation of the present state of our knowledge justify this encroachment upon an adjoining domain. In discussing this group we shall not omit to describe the numerous pathological alterations in the nerves which have been brought to light by inquiries undertaken during the last few years, and a systematic description of these, so far as they can be recognized from the symptoms accompanying them, as definite forms of disease, will form the subject of the second part.

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FUNCTIONAL DISEASES (NEUROSES) OF THE PERIPHERAL NERVES.

Romberg, Lehrbuch d. Nervenkrankheiten, 2. u. 3. Aufl., Bd. I.—*Hasse*, Krankheiten des Nervensyst., 2. Aufl., 1869.—*M. Rosenthal*, Handb. d. Diagnost. u. Ther. der Nervenkrankheiten, 1870.—*A. Eulenburg*, Lehrb. d. functionell. Nervenkrankh., 1871.—*Hammond*, Diseases of the Nervous System, 3d edit., 1873.—*Benedict*, Electrotherapie, 1868.—Nervenpathologie, etc., 1874.

Consult also the bibliographical lists given in the following chapters and in the manuals of electro-therapeutics.

NEUROSES OF THE SENSORY NERVES.

Functional disorders of the centripetally conducting, or, as they are commonly termed, sensory nerves, constitute the essential feature of the so-called *sensory neuroses*. All the pathological phenomena occurring either in the peripheral or central terminal organs of centripetally conducting nerves, providing they attain a certain degree of independence, represent either partial or complete sensory neuroses. It is obvious, on general pathological grounds, that all such disturbances must owe their origin to the existence of conditions similar to those that accompany the ordinary functional activity of the sensory apparatus. A glance at the normal processes that take place in sensation shows at once in what way those pathological disturbances of sensation arise which essentially constitute sensory neuroses. Every sensation, as a general rule, results from a change caused by an external agent or stimulus acting on some excitable part of the sensory apparatus which it thereby excites. As a rule it is the peripheral terminal organs of the sensory nerves that are excited, and in which the excitation commences. This state of excitation is conducted through centripetal nerves to the nervous central organs, in accordance with the law of isolated conduction. It is then transmitted to the central terminal apparatus of the centripetal nerves, inducing in them those changes which constitute the basis of *conscious sensation.* We possess no precise knowledge in regard to the more recondite details of these processes, nor even in regard to the part of the brain in which they occur.

The connections of the centripetal nerves with the central sensory apparatus of the brain are not the only ones that they form. Others exist which are for the most part situated in the central organs, and are established partly with other portions of the sensory apparatus, and partly with centrifugal, and especially with motor, vaso-motor, and secretory nerves. We have here, therefore, examples of the processes, that have been more or less completely investigated by physiologists, of the radiation of sensations (associated sensations), reflex movements and reflex actions exerted on vaso-motor nerves and upon the secreting organs. These connections seem indeed adapted to enable an influence to be exerted on still wider and more remote nerve territories, so that centripetally running excitations may occasion modifications of the cardiac and respiratory actions, and of the process of digestion. Little as we know of the anatomical means by which these processes are accomplished, yet it is certain that they play a great part in the pathology of the sensory system, as further observations will show.

The numberless sensations of which we are thus made conscious, present the utmost variety in regard to their quality and strength. The *qualitative modification* of the sensations can either be determined by the qualitative anatomical differences of the terminal apparatus to which the stimulus is applied—from which it follows that certain sensory apparatus can only communicate quite definite sensations, and are only excitable by very special stimuli, as in the case of the higher organs of sense, and the tactile organs;—or it is determined by the peculiar nature of the stimulus, whence it results that the same sensory apparatus can be excited in a different way by different stimuli, and can supply information to the conscious mind of qualitative differences in the stimulus applied.

The quantitative modifications of the sensations, as of their intensity and strength, can be determined either by the strength of the stimulus or by the excitability of the sensory apparatus. The greater this excitability at a given moment, the more readily is the sensory apparatus affected by the stimulus, and by so much the greater is the acuteness of the sensation; on the other hand, the lower the excitability, the feebler is the intensity of the sensation called forth by a given stimulus. Under all ordinary circumstances our sensory apparatus preserves a medium degree of excitability, so that from the strength of our sensations we can form some idea of the intensity of the stimulus. Under pathological conditions this may be different.

The pathological modifications of the sensory processes, which constitute the essence of sensory neuroses, may be referred to the same conditions. In pathological states we constantly meet with *anomalies in the intensity or strength of the sensations.* These may be occasioned, 1. By a *change in the excitability of the sensory apparatus*, which under physiological conditions remains nearly stationary.

We meet with, *a*, an *exaltation of excitability*, an increased sensitiveness to stimulation, so that slight stimuli induce very active sensory processes, and rapidly induce the higher degrees of sensation until actual pain is felt. This state, in which it is obvious that the molecular mobility in the sensory apparatus is exalted, is commonly termed *hyperæsthesia*, and it may affect various parts of the sensory apparatus; as, on the one hand, the peripheral terminal apparatus, which then acquires a greater mobility of its molecules, and a greater degree of excitability, and reacts with disproportionate energy to every irritant, or some part of the conducting fibres, by virtue of which an excitation of ordinary strength becomes augmented in intensity, and is conducted from the affected part onwards to the central organ, as a stronger stimulus; or, lastly, a similar condition of excitability may exist in the central sensory apparatus, so that excitations, conducted from the periphery, produce an unnaturally active change in the consciousness, or, in other words, induce an abnormally intense sensation; examples of all these modes can easily be drawn from daily experience at the bedside.

b. In other cases, however, we meet with *diminished excita-*

bility of the sensory apparatus, so that slight stimuli either pass unperceived, or are felt but indistinctly, while powerful stimuli are felt only feebly, or, in high degrees of the affection, are not perceived at all. This is the condition termed *anæsthesia*, in which we must admit great inertia or even complete immobility of the molecules of the sensory apparatus. And this, like hyperæsthesia, may occur in different parts of the sensory apparatus. The peripheral terminal apparatus may be in a state of diminished excitability or of total inexcitability, and thus the establishment of the excitation may be rendered difficult or impossible; or obstacles to the transmission of the excitation may exist in the conductory fibres, so that conduction is rendered difficult and slow, or is altogether arrested; or, lastly, the centric sensory apparatus may be less excitable, and thus cause only a slight change in the consciousness compared with the strength of the stimulus. These conditions are also all met with in diseases of the nervous system, though no doubt with varying degrees of frequency, the so-called "anæsthesia of conduction" being most common.

Anomalies in the intensity or acuteness of sensation can also be determined by

2. *Variations in the intensity of the stimulus*, and most frequently by *abnormally powerful stimuli.* These induce great changes in the sensory apparatus, which may gradually rise to the most intense pain. Such abnormally strong excitations are best known as intense external stimuli, wounds, chemical actions, heat, cold, etc.; but they may also arise in the organism itself, and there act on the sensory apparatus; they then cause sensations (pain and the like), which are usually termed *spontaneous*, in order to distinguish them from those caused by external excitants; examples of such internal stimuli may be found in inflammation, hyperæmia, pressure of new-growths, disturbances of nutrition, and the like. Whether the diminished action of normal stimuli—abnormally weak stimuli—can occasion pathological changes of the sensory processes must be regarded as doubtful.

The pathological changes hitherto mentioned are, however, not the only ones which may be observed in the sensory apparatus; for *anomalies in the quality of the sensation* are also met with; new and unusual sensations are then experienced for which no adequate cause exists, sensations which present indeed a certain degree of similarity with many of those that are produced by external stimuli, and are named accordingly, but which arise spontaneously from internal stimuli. Amongst these are formication, itching, numbness, furry feeling (Pelzigsein), burning and the like, which the patient is often unable to describe more exactly, but which, for the most part, differ remarkably from ordinary sensations. It is advantageous to apply the general term *parœsthesia* to these peculiar and in pathological states not very uncommon sensations. It is highly probable that such sensations originate in various parts of the sensory apparatus, either peripherically, centrically, or in some part of the conducting paths; very little is, however, known either in regard to their cause or their mode of origin. Abnormal internal stimuli are almost always involved, and it is probable that such paræsthesiæ are most commonly produced by trophic disturbances or by modifications of the circulation, or by certain mechanical and thermic influences.

It may suffice to have thus briefly referred to the general pathological ideas associated with hyperæsthesia, anæsthesia, and paræsthesia, and to have characterized them as symptoms of the sensory apparatus, that may appear in the most diverse forms of nervous disease, and may frequently occur as one of the symptoms in sensory neuroses.[1] We must in like manner here limit ourselves to a very short exposition of certain physiological laws governing the normal sensory processes, which, preserving their importance, frequently afford us welcome information in pathological conditions. These laws are: the *law of*

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[1] *A. Eulenburg* in his text-book has endeavored to subdivide the various forms of hyperæsthesia and anæsthesia, in reference to differences in the quality of the sensations (sensory impressions and common sensation) by sharp points of distinction. Thus he designates the corresponding anomalies of common sensation as hyperalgia, analgia, and paralgia; the anomalies of pain as hyperalgesia and analgesia, those of touch as hyperpselaphesia and apselaphesia (after Eigenbrodt), etc. These terms, on account of the difficulty of their terminology, will be accepted only with slowness, and the distinction between hyperalgia and hyperalgesia is not sufficiently well founded in an etymological point of view.

isolated conduction, according to which any excitation applied to a centripetal fibre, is conducted by this alone to the central apparatus, and is not transmitted to other adjoining fibres; the *law of excentric projection*, according to which excitations affecting either the central sensory apparatus itself, or any part of the centripetal conducting fibres, are referred by the mind to the peripheric extremities of the conducting fibre, from whence experience has shown that physiological stimuli usually proceed (examples of this are found in the cases so frequently cited where irritations of the nerve stumps of amputated limbs have been referred to their peripheric extremities, and cases where irritations of the centripetally conducting fibres of the spinal cord or brain have been referred to the cutaneous surface of the limbs, etc.); the *law of associated sensation or irradiation*, according to which irritation of centripetal fibres in the central organ (probably through the intervention of ganglion cells in the gray substance) can be transferred to other centripetal paths, and thus excite co-sensations which are again referred by the mind, in accordance with the laws of excentric projection, to the periphery. According to this law the peripheral excitation of a centripetal fibre may be accompanied by a sensation in a more or less distant part of the periphery; as an example, the occurrence of an "irradiated" pain in the back, on strong irritation in the region of our sciatic nerve, may be mentioned. Lastly, *the law of reflex action*, according to which irritations of centripetal fibres in the central organs (probably owing to the intervention of ganglionic apparatus in the gray substance) may be transferred to centrifugal fibres—motor, vaso-motor, and secretory nerves—and exhibit their special action at the periphery; these reflex actions take place according to laws that are accurately laid down in physiological works, and play a great part in the phenomena accompanying sensory neuroses.

The impressions communicated by the sensory apparatus are, for the most part, mentally referred to changes in the outer world, and all the changes of consciousness induced by external agents are termed *objective* sensations. But the sensory apparatus is also affected by impressions which are directly called forth by changes in our bodies, and which are termed *feelings* or *subjective* sensations. The sum of all these feelings is known as *common sensation.* All our sensations are really excited by changes of the sensory apparatus, but it is only through a part of these that they reach our consciousness. We have thus in the higher sensory organs, as the eye and ear, objective sensations alone, that is to say, we refer stimuli affecting them to the outer world, whilst the changes they produce in the sensory organs themselves are not in any way felt. It is only in quite special conditions, or with extraordinarily violent influences that subjective feelings are excited, which are referred to a change in the sensory apparatus, and we then say that something "effects the eye or the ear, pleasantly or unpleasantly." Subjective symptoms of this kind are felt much more vividly and more frequently in those organs of sense in which we are accustomed to refer the place of action of the stimulus directly to the peripheral terminal organs, as in the case of the sensations communicated by the skin or tongue. All sensations are in these instances accompanied by a feeling of change of the body itself, and subjective and objective sensations are not always sharply discriminated from each other by the mind. In these instances sometimes the objective and sometimes the subjective nature of the sensation is more distinctly experienced, according to the strength and the kind of the stimulus, and according to the degree of the excitability and sensitiveness of the part. Certain kinds of stimuli, and certain degrees of their intensity, on the one hand, and on the other certain sensory organs (the genital nerves for example) chiefly communicate common sensations. Under this term of common sensation we usually include tickling, itching, pain, shuddering, sexual pleasure, hunger and thirst, etc. Such sensations play an important part in the pathology of the nervous system, and those proceeding from the sensory organs in the skin, the so-called cutaneous sensations, on account of their frequency and strongly marked characters in disease, are of great practical importance and require special attention.

The most general and the most frequently observed common sensation, which it would appear may be excited in almost all sensory organs, is *pain*. Pain is so important a symptom of disease in practice, and plays so prominent a part in the sensory neuroses, that it may be advisable to give a short description of it here.

In many diseases pain is the first, and in some it is the only symptom. For a large number of patients it is the most difficult to bear, and is hence for them the most important symptom; nor is it of less importance to the physician, since it frequently affords him the means of determining the position, the nature, and the intensity of the disease. All possible degrees and modifications of pain may be felt, from the slightest unpleasant sensation to the most intense, almost insufferable agony, rendering the patient frantic, driving him to desperation, and even to suicide. It is only rarely, however, that definite conclusions in respect to the nature or the grade of the disease occasioning it can be drawn from the varying intensity of pain, because it depends too much upon the character of the patient, his excitability, and his psychical constitution; we do not even possess any objective measure for the degree of pain, and can only approximatively estimate it from the statements of the patient, after making due allowance for all the above circumstances.

The varieties of pain are as manifold as its degrees, and we are accustomed to distinguish several forms, as burning, tearing, boring, stabbing, tensive, dull, etc. As we shall immediately point out, the various kinds of pain are not different kinds of common sensation, but the pain is always the same modification of consciousness, which, however, may be accompanied by various coincident sensations that give it its local characters. Thus when the pain is burning we have coincidently a sensation of heat; in tensive pain, the sensation of some external agent stretching the sensory apparatus over a large surface; in stabbing pain, the feeling as if one or several nerve-fibres were irritated by a pointed body, etc. Spontaneous pains produced by internal stimuli are mentally compared with those that are called forth by well-known external stimuli, or by physiological processes (as for example by labor pains), and which induce similar co-sensations, and are named accordingly. Lastly, there seems to be reason for believing that the disposition of the sensory

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nerve-paths in certain parts, their pathological excitation, and the like, are not without influence upon the mode in which sensations of pain are perceived and estimated. It is here again very difficult, on account of our ignorance of the more delicate processes, to draw definite conclusions in regard to the nature of the disease from the kind of pain that may be present. Experience has at the same time taught us that in certain diseases certain kinds of pain occur with special frequency, so that they possess a certain importance in establishing the diagnosis, as is the case, for example, in the stabbing pain of pleurisy, the racking pain of rheumatism, the boring pain of neuralgia, and the throbbing pain of many forms of inflammation.

Most organs and tissues of the body are capable of becoming the seat of pain, though with different facility and frequency. Some few, as cartilage, are probably never the seat of pain; others, as muscles, gland substances, and certain parts of the central nervous system, appear to be normally insensible, and only become painful under certain pathological conditions; others again, as the skin, the serous and synovial membranes, many parts of the mucous membranes, and the sensory nerves, are the seat of the most acute pain in disease. Those organs and tissues which normally furnish the most distinct and frequent sensations, are just those in which, under pathological conditions, pain most readily arises, and in which, from long physiological experience, we can most certainly localize the pain. Deception does not readily occur in regard to the seat of a pain either in the skin or in the mucous membranes communicating with the surface of the body, or in the joints; but, on the other hand, much uncertainty often prevails in regard to the localization of pain arising in organs that are physiologically insensible, and the precise seat of pain is often correctly ascertained only after much experience, and by the help of numerous artificial aids and artifices. This is not so much the case in pain communicated by the great sets of sensory nerves (true nervous pains, neuralgia), in which at least the physician can correctly and readily localize the pain by means of his anatomical knowledge.

Many authors have endeavored to give a precise definition of pain, but the widely differing statements that have been made show that there is some difficulty in doing so, nor is this in any way surprising. Both physiologists and physicians, however, are for the most part agreed that pain is a common sensation that belongs not to a peculiar kind, but only to a certain degree of sensation. Valentin[1] applies the term pain to those "sensory impressions which, on account of their too great intensity, become disagreeable." Wundt[2] defines pain to be "a feeling that accompanies all powerful or intense stimuli," and A. Eulenburg[3] understands pain to be "a gradual increase of the feeling that accompanies every sensory process." We hold that every increase of ordinary sensory stimuli is capable of producing pain, as soon as it attains a certain intensity. Every excitation, the intensity of which exceeds certain limits, every molecular change of the centripetal series, induced by an abnormally strong stimulus, is perceived as pain. Very simple experiments, as, for example, pressure or temperature gradually increased till pain is produced, show that with very gradual increase in the strength of the stimulus, a limit is at length reached beyond which the excitory process is accompanied by pain, yet no sharp line of demarcation can be traced defining the point at which the sensation of pressure or temperature ceases, and the sensation of pain commences. The simplest explanation accordingly seems to be that pain is the reaction of the sensorium to a certain degree of excitation, and we at present see no ground for regarding pathological pain as being essentially different in origin from that which can be produced by simple physiological experiment. By this, however, it is not meant to imply that the requisite intensity of the excitory process always occurs in pathological conditions in consequence of an increase in the intensity of the stimulus, for it may also originate in other conditions, and probably does so in the greater number of pathological cases.

After what has been said it is only reasonable to expect that at times impressions of painful intensity are transmitted by all centripetal nerves—those of the cerebro-spinal system. No doubt, as a rule, those nerves that are specially regarded as sensory nerves, as the nerves conducting tactile impression and common sensation, are those which conduct pain; but it must not be misunderstood, that true sensations of pain can be communicated through the nerves of special sense by increasing their specific energy and the specific stimuli affecting them. Thus it happens in particular, both in the case of the eye and in that of the ear, that intense light and loud sounds (detonation, inharmonious notes, grating and whistling sounds, and the like) produce sensory impressions which, as is commonly said, "pain the eye or the ear."

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[1] *Valentin*, Physiol. Pathol. der Nerven, Band I., p. 240.

[2] *Wundt*, Lehrbuch der Physiologie des Menschen, 1874, p. 503.

[3] *A. Eulenburg*, Functionelle Nervenkrankheiten, p. 31.

Pain, therefore, as communicated by the various centripetal nerves has no specific quality. It is certainly in a measure a *new* sensation which is first experienced when the excitation attains a certain intensity, and represents a perfectly definite change in the sensorium, accompanied by the conception or idea of the unpleasant affection of the "ego" we designate as pain. This new sensation is not, however, in any way to be regarded as the consequence of a new species of excitation in the centripetal nerves, but only as the expression of a definite reaction of the sensorium to all excitations of a certain strength. On this account powerful stimuli, however various they may be, induce one sensation only—pain—in the different centripetal nerves. But with this sensation of pain there may also be mingled, by a coincident specific excitation of the sensory apparatus, a localized impression from which we are to some extent capable of deducing the cause and nature of the pain. With the aid of our tactile sensibility, our sense of temperature and of locality, we can readily distinguish the pain of a hot body from that of the prick of a needle, or from that produced by a cut or by pressure. And from the intermixture of such coincident sensations, from the localization of the excitation and its varieties, it is obvious that we can also characterize the idiopathic pains, that occur in disease, as burning, tearing, stabbing, boring, tensive, and the like. Of course no information is thus conveyed of the proper causes, nor of the various forms of the excitation; we do not know what really causes a pain resulting from disease to be burning or stabbing, but we are here only comparing sensations within the sensorium with those that depend on known causes. It must consequently be regarded as a fundamental requisite for the occurrence of pain that, besides excitability of the peripheric terminal apparatus, and healthiness of the conducting fibres, the central sensory apparatus should possess its normal functional activity. The present state of cerebral physiology does not permit us to say more; yet it may be reasonably maintained that the condition of the gray substance is of special importance. In accordance with this, physiological experiments seem also to show that the gray substance of the spinal cord is of extreme importance in regard to the occurrence of painful sensations. Schiff[1] long ago discovered, and his later researches have corroborated his statements, that tactile sensations are conducted to the brain through the posterior white columns of the spinal cord, whilst the paths for the conduction of pain are situated in the gray substance of the spinal cord. Whether such a separation of the paths for pain and tactile sensations really exists in the spinal cord, or whether, by violent pain-producing stimuli, the change essential for the production of a sensation of pain, which is different from conduction, is called forth in the gray substance of the spinal cord, must remain at present undecided. The fact discovered by Schiff possesses at the same time great value in pathology.

After what has been said, it is easy to point out under what conditions pain occurs; it is wherever the intensity of the centripetal excitation oversteps a certain limit. This may be attained in two ways: on the one hand, by the increase in the intensity of the stimulus, and to this belong the simplest cases of the production of pain by wounds, caustics, extremes of temperature, and the like; and secondly, by the increase in the excitability of the sensory apparatus (hyperæsthesia), so that weak stimuli produce pain. The last condition is much the most frequent in the so-called idiopathic pains occurring under pathological conditions.

Although we can thus sharply define the conditions requisite for the production of pain, we are still unable to explain accurately in what way pain actually arises in the various forms of disease—how, for example, in inflammation, catarrh, cramp, neuralgia, etc., such powerful sensory stimuli are produced that pain is felt. In rare cases it is due to an increase in the strength of the stimuli, and coarse mechanical conditions are then usually in operation; but in the great majority of cases we are compelled to admit the occurrence of molecular changes, such as trophic disturbances in the sensory apparatus, which cause increased excitability or augmented molecular irritability, so that various slight stimuli, as those of the physiological movements of the body, the circulation of the blood, the ordinary strains made upon the tissues, are capable of augmenting the irritation till pain is produced. But we are not at present in a position to give any satisfactory account of the mode in which these trophic disturbances occur in disease, how they are developed from their causes, and how they are to be exactly defined.

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[1] *Schiff*, Lehrbuch der Physiologie des Menschen. Lahr, 1859.

It hence appears that at present all discussion in regard to the essence of pain is idle. So long as we are in the dark in regard to the finer molecular changes that accompany it, and in regard to the psychological questions in connection with it, no satisfactory theory of pain can be given. It is unnecessary to enter into a discussion whether pain originates in augmented or in diminished functional activity of the centripetal nerves, although, according to our views, it cannot remain doubtful that in the production of pain an increased excitation originates in these paths. The state and the mode of reaction of consciousness itself, or of the central sensory apparatus, still constitute the chief condition in all instances, whilst the peripheral apparatus and the conducting fibres, when pain is experienced, have nothing further to do than to receive the irritation caused by augmented intensity of the stimulus, quite in the ordinary manner, and conduct it to the cerebrum. The hypothesis most worthy of discussion appears to be that advocated with so much ingenuity by Griesinger,[1] which is to the effect that the essence of pain lies in "a disturbance of the organization" of the nerve at some point in its course. In regard to this it may be remarked that molecular changes must here no doubt be present, though not discernible by us, and since we are now accustomed to consider every excitation as accompanied by a molecular change, and since no sharply defined line of demarcation can be drawn between these physiological molecular changes and pain-producing "disturbances of the organization," no step in advance is made by this hypothesis. It is, moreover, inadequate to account for a large number of painful impressions, in which it is impossible to admit a "disturbance of the organization" which is in any way persistent. The precise definition of pain must thus be postponed to a subsequent period. The points which specially relate to pain in the sensory neuroses, and which are here deserving of particular attention, hold also for pain in general, and have already been given. In discussing the several forms of disease, the special characters that the pain accompanying them may happen to have will be fully described. In inflammatory and analogous peripheral pains we have to do for the most part with irritation of the terminal expansions of the sensory nerves, whilst in the neuroses we are chiefly concerned with irritations of the nerves external to and within the central organs, as well as with the central terminal apparatus, and it is upon this that most of the peculiarities of pain in neuroses depend. The further discussion of this point would here be superfluous. It is self-evident that both for the sensations of pain and for their causes the above-mentioned physiological laws governing the sensory apparatus preserve their supremacy, and we shall hereafter meet frequently with phenomena of irradiation, of excentric projection, and of reflex action in the painful affections of sensory nerves.

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[1] Archiv für physiol. Medic., Band I., 1843.

1. Neuralgia in General.

Neuralgia, as a special form of disease, has not been very clearly described by the older physicians, and it has hence been inferred that in former times it was comparatively rare. Nothing definite is stated in regard to it by Hippocrates. Aretæus is the first who gives a recognizable description of it, though without any accurate insight into its etiology, and the same may be said of Galen and Paulus Ægineta; the Arabians, Rhazes, Avicenna, Albucasis, were, however, there can be no doubt, intimately acquainted with it. In the middle

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ages, and even up to the middle of the eighteenth century, only imperfect description and recognition of neuralgia existed. The first correct explanation of neuralgia as a painful affection of the nerves dates from André, who in 1756 first described "Tic douloureux." After this, numerous facts and observations were accumulated, and special cases of facialgia and sciatica aroused the interest of physicians. The literature of the subject from this time forward is extremely rich, and only a few of the principal works are here appended (for a complete bibliography, see Bretschneider, l. c.).

De Haën, Ratio medendi, 1764.—*Domen. Cotugno*, Comment. de ischiade nervosa. Neapol., 1764.—*J. Fothergill*, Med. Observat. and Inquir., T. IV. and V., 1768 and 1773.—*Chaussier*, Table synoptique de la névralgie, etc. Paris, 1803.—*Jos. Frank*, Univ. praxeos med. præcept. Lips., 1821.—*Martinet*, Revue franç. et étrang. Paris, 1828.—*Ch. Bell*, The Nerv. Syst. of the Human Body. Lond., 1830. Dtsch. v. Romberg. Berlin, 1832.—*Piorry*, Gaz. médic., 1833.—*B. Brodie*, Lect. Illustr. of Certain Local Nervous Affections. Lond., 1837.—*Allier*, Journ. de Méd. et de Chir., 1838.—*Eisenmann*, Die Krankheitsfam. Rheuma, 1841.—*C. James*, Des névralgies et de leur traitement. Paris, 1841.—*Valleix*, Traité des névralgies ou affect. doul. des nerfs. Paris, 1841. Deutsch v. Gruner. 1852. (The references to this work in the following pages are made from the German edition.)—*Bretschneider*, Path. u. Therap. d. äussern Neuralgien, 1847.—*Türck*, Beitr. z. Lehre v. d. Hyperästhesie and Anästhesie, Zeitschr. d. Ges. d. Wien. Aerzte, 1850.—*Henle*, Handb. d. ration. Pathol., II., 2, 1853.—*Parson*, Neuralgia; its History, Nature, and Treatment, Amer. Journ. of Med. Sc., XXVIII., 1854.—*A. Notta*, Mém. sur les lésions fonctionnelles, qui sont sous la dépendance de la névralgie, Arch. génér., 1854.—*Baerensprung*, Ueber Zoster., Annal. d. Charitékrankenh., IX., X., XI., 1861–1863.—*Vanlair*, Ueber Neuralgien, Journ. de Med. de Brux., 1865.—*Romberg*, Kritik der Valleixschen. Schmerzpunkte Arch. f. Psych. u. Nervenkr., I., 1867.—*Nothnagel*, Troph. Störungen bei Neuralgien, ibid. II., 1869.—*Nothnagel*, Schmerz und cutane Sensibilitätsstörung, Virch. Arch., Band 54, 1872.—*Anstie*, Neuralgia and Diseases that Resemble it, 1872.—Much may be found in the text-books of Special Pathology and of Nervous Diseases, as well as in the manuals of *Puchelt*, *Canstatt*, *Wunderlich*, *Trousseau*, *Lebert*, *Niemeyer*, *Kunze*; also in *Romberg*, Lehrb. der Nervenkrankh., 1 Aufl., 1840; 3 Aufl., 1853.—*Hasse*, Krankh. des Nervensystems, 2. Aufl., 1869.—*Eulenburg A.*, Functionelle Nervenkrankheiten, 1871.—*Rosenthal, M.*, Diagnostik u. Therapie d. Nervenkr., 1869.—In regard to the treatment of neuralgia, see *A. Eulenburg*, Hypodermat. Injection, 1867, 2. Aufl. (the bibliography is given very fully in this work).—See also the text-books of electro-therapeutics, which besides the therapeutics contain also much information in regard to the pathology of neuralgia: *Duchenne*, *M. Meyer*, *Remak*, *Frommhold*, *Althaus*, *M. Rosenthal*, *Beard* and *Rockwell*, *Onimus* et *Legros*; also *W. Leube*, Beitr. z. Beh. d. Neuralg. mit dem inducirt., Strom. Diss. Tübing., 1862.—*E. Flies*, Electrother. Mittheil. im Gebiet. d. Neuralgien, Allg. med. Centralz., 1862.—*Erb*, Galvanotherap. Mittheilungen, Arch. f. klin. Med., III., 1867.—*Berger*, Electr. Behandlung des Tic doulour. und Hemicranie, Berl. klin. Woch., 1871, No. 2.—In reference to the excision of nerves, consult the works of *Bruns*, *Wagner*, *v. Nussbaum*, *Podratzky*, *Patruban*, *Schuh*, and others mentioned in the bibliography at the beginning of the chapter on Neuralgia of the Trigeminus.

Definition.—The term *neuralgia* signifies a disease of the sensory nervous apparatus, the chief and most important symptom of which is pain.[1] The pain appears to be localized in particular nerve trunks or branches, with all their ramifications; it is characterized by its remarkable acuteness, and is either intermitting, or at least undergoes very well-marked remissions. It is felt for the most part not in the periphery alone, but throughout the whole course of the affected nerve, or at least at several points of this course. The pain is accompanied by secondary phenomena of a motor, vaso-motor, secretory, and trophic nature, which are partly attributable to an augmentation of the physiological relations existing between the sensory and the above-named nerves, and are partly concomitant effects of the same conditions that occasion the pain. The general health, in relation to the great subjective discomfort, is disturbed to a remarkably slight extent. In the greater number of cases the most careful examination furnishes no evidence of palpable organic lesions, apart from a few points, at various parts of the affected nerve, that are sensitive to pressure.

It thus appears that no concise or complete definition of the term "neuralgia" can at present be given. The most important and fundamental facts required to enable us to make such a definition are wanting, that is to say, we have no precise knowledge of the finer anatomico-pathological changes in the parts, or of the relation of these changes to the causes of neuralgia; moreover, it is not practicable to discriminate those definite forms of neuralgia that are known either as essential or as idiopathic,—of which latter we know the least—from the symptomatic ones caused by coarse anatomical changes; such a division is simply impossible clinically, inasmuch as the phenomena of the two forms are identical. We shall, indeed, endeavor to show, in treating of the pathogenesis of neuralgia, that such a division in no way corresponds to the essential nature of neuralgia.

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[1] As pain in general is only communicated by the sensory nervous apparatus, it has a certain claim to the designation "neuralgia" (from νεῦρον and ἄλγος). Custom has, however, applied the term to the forms of disease now under consideration; we speak of muscular pain, pain in the bones, with the same right.

The following points may be regarded as particularly characteristic of neuralgia: the limitation of the pain to a definite trunk or branch of a nerve, and its ramifications; shooting pains along the course of the nerve and their spontaneous recurrence without any apparent external cause; the paroxysmal character of the pain, which presents distinct intermissions or remissions; the absence of disease in other organs of the part (except those of the nervous system itself), and the absence of any remarkable disturbance of the general health. On the other hand, amongst the non-characteristic features may be enumerated the occurrence of pain on light contact, and its disappearance when strong pressure is made, a symptom that has been included by Bretschneider and others in the definition of neuralgia; improperly so, for it is absent in many instances of true neuralgia, and may be present in other forms of disease.

Pain, then, is the most prominent symptom in neuralgia, not only as regards the feelings of the patient, but as an object of clinical investigation and treatment. It has been often overlooked that the above-mentioned secondary symptoms belong properly to the physiognomy of the disease, and that in some cases they even form essential features of it. As we examine the affection more closely, we shall obtain an idea of their frequency, and shall have to point out in what way an exact knowledge of their pathogenesis can be applied in drawing accurate and useful conclusions in regard to the neuralgia. For, as might be expected, very definite conclusions as to the seat of the neuralgic affections may be drawn from a careful consideration of the presence and nature of such associated phenomena.

Neuralgia must in the present state of our knowledge be regarded as a symptom. In many cases distinct anatomical changes are perceptible, but the mode in which they cause the neuralgia is not very clear, since the most diverse anatomical changes may produce the same symptoms. In the great majority of cases no material changes, at least with our present means of research, are demonstrable in the nerves affected; it would appear, therefore, that we are quite justified in maintaining the symptomatic unity of neuralgia, and the more so since it offers in the most different cases very much that is characteristic and accordant, in regard to mode of appearance, course, etc.

Neuralgia is by no means identical with *hyperæsthesia* of the sensory nerves, and on this account it does not appear to be correct to include it under the head of hyperæsthesia, as Romberg and his followers have done. On the other hand, it certainly is not sharply defined from hyperæsthesia. In hyperæsthesia augmented excitability may exist without pain, providing no irritation affect the preternaturally sensitive nerve. We in fact may be said to recognize hyperæsthesia by the augmented reaction which the nerve exhibits to the action of external stimuli. In neuralgia, on the other hand, pain exists apparently without the action of any external stimulus, and it is in many cases due to abnormally strong internal stimuli. It must be admitted, however, that increased excitability frequently exists in nerves affected with neuralgia, which facilitates the supervention of pain, and increases its intensity. Every case of hyperæsthesia, therefore, does not appear under the form of a neuralgia, nor can every case of neuralgia be traced back to an existing hyperæsthesia; at the same time both phenomena are frequently coincidently present in many forms of the disease.

Etiology.

Neuralgiæ are undoubtedly amongst the most common of neuroses. They occur in all classes of society, at all periods of life, and at all seasons of the year. In many cases they are referable to very definite and well-recognized injuries. But in just as many others the causes are obscure, and can only be guessed at; in all cases, however, the more intimate relations between cause and effect are hypothetical.

Here, as elsewhere in etiology, it is necessary to distinguish between remote predisposing and proximate exciting causes.

The latter, if they are of moderate intensity, act only on those who are predisposed; but if they are very intense they may affect the perfectly healthy. The former, or predisposing causes, may be so intense that agencies inappreciable by us may suffice to produce neuralgia.

a. *Predisposition.*

Of all the circumstances now under consideration none offer greater interest than the *neuropathic predisposition*, which has been established and very carefully investigated of late years, and which plays a very important part in the etiology of a great number of neuroses. Amongst these we may enumerate the psychoses, epilepsy, chorea, hysteria, and the like. By this phrase is understood a pathological constitution affecting the functional activity of the nervous system (Griesinger), by virtue of which those who are thus constituted manifest throughout life the most varied pathological symptoms in regard to sensory, motor, or psychical processes. No one has as yet been able to show in what this peculiar anomaly consists, and while some console themselves with the hypotheses of "delicate trophic disturbances" or "modifications of molecular arrangement," without thereby getting any nearer to the facts, we must rest satisfied with the fact that such constitutional neuropathies really exist, and that they play an important part in the history of neuralgia. They may be congenital and proceed from the parents, and hence be *hereditary*, or they may be *acquired* in the course of life as a consequence of the most diverse injurious influences. Of special importance is the *hereditary neuropathic predisposition*, that unfortunate condition which forms the inheritance of so many families, in which the most diverse forms of neurosis are, so to speak, innate, and propagate themselves from one generation to another, sometimes affecting chiefly the psychical, sometimes the sensory, and sometimes the motor and vaso-motor regions of the nervous system. To it many forms of neuralgia owe their origin. Anstie, who has paid particular attention to this neuropathic hereditary predisposition, attributes to it not only a predisposition to psychoses, epilepsy, chorea, hysteria, paralysis, etc., but also to phthisis, and states that in 83 cases of neuralgia investigated with regard to this point, he found there were 71 in which such a family predisposition existed, and of these 53 cases occurred in neuropathic, and 18 in phthisical families. In such cases the coincidence of neuralgia with other neuroses, or its alternation with epilepsy, various psychoses, migraine, etc., is not unfrequently observed. This hereditary origin of neuralgia is of course most obvious in the not unfrequent cases where there is a direct hereditary transmission from the parents to the children, where the father or mother has suffered from neuralgia and the child has likewise been subject to it. Anstie found that this direct descent of the disease occurred in 24 cases in 100, whilst in 58 it did not occur, and in the remaining 18 no positive information could be obtained. It would be well if in future researches more attention were directed to the point. The neuralgiæ which so commonly form one of the symptoms of hysteria, and more rarely of hypochondria, are very closely allied with those that are attributable to a neuropathic disposition.

The period of life is an influence which must not be underestimated as a predisposing cause of neuralgia. In childhood there is but little predisposition to it, though cases are undoubtedly occasionally met with. On the other hand, in adolescence, especially at the period of sexual development, a great number of neuralgic affections originate. They are most common, however, in middle life, between the ages of twenty and fifty. Even in more advanced life a strong predisposition to them exists, and the numbers reported are of so much the greater significance if we take into consideration their relative proportion to the total number of individuals then alive. This predisposition of advanced age to neuralgic disease depends in part upon the powerful influence of the climacterium upon the nervous system, and in part on the more or less early senile degeneration of the tissues, especially of the arteries and nerves.

Comparative estimates in regard to the occurrence of neuralgia at different ages have been made by various authors. The tables of Valleix and Eulenburg are here appended, together with those of 146 cases that have fallen under my own care.

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Period of Life.	Valleix.	Eulenburg.	Erb.	Total.
Up to 10 years,	2	6	..	3
10–20 "	22		14	41
20–30 "	68	19	40	127
30–40 "	67	33	39	139
40–50 "	64	23	29	116
50–60 "	47	14	14	75
60–70 "	21	6	9	36
70–80 "	5	..	1	6
	296	101	146	543

The proportionate numbers are somewhat different if the neuralgiæ of particular regions are examined (see Bretschneider, l. c., p. 216 et seq.).

From the statistics at present at our command no very definite conclusions can be drawn in regard to the influence of *sex* upon the frequency of neuralgia in general. It is usually considered that women are more predisposed to it than men; thus Valleix found that in 469 cases 218 were men and 251 were women; Eulenburg, that in 106 cases 30 were men and 76 women; Anstie, that in 100 cases 68 were women and 32 men; whilst I have found in 146 cases 84 men and only 62 women.[1] The differences in these statements depend apparently upon the accidents of practice. There can be no doubt, however, that sex makes a considerable difference in regard to the occurrence of certain forms of neuralgia. Thus all observers agree that neuralgia of the fifth nerve is more frequent in females than in males, whilst in sciatica the opposite occurs, but even here perhaps there may be less owing to actual difference of sex than to the contingencies of external influences.

The *sexual periods of life* are of great importance in regard to the development of neuralgia. The profound influences exerted upon the nervous system by the genital organs during and after puberty, the great revolution that is effected in the entire organism, owing to the awakening of the sexual activity, the extreme irritation of the nervous system from overpowering desire, and the exhaustion from over-frequent or unnatural gratification, are only too fruitful causes of those changes in the nutrition of the nervous system which occasion the neuropathic predisposition. Thus we see that the period of puberty and of the grand climacteric, that the occurrence of the menses, that pregnancy and the puerperal period, are particularly fruitful in the production of neuralgic affections, and we see, too, that sexual excesses, and especially the vice of masturbation so frequently practised by both sexes in the present day, are often punished by the development of neuralgic complaints. On this subject Anstie makes some practical observations that are well worth consideration, and points out, amongst other things, that it is not only the natural or unnatural excessive yielding to the sexual passion that exerts so deleterious an influence, but that the unconscious excitations, even in chaste and pure-minded persons, which arise from the sexual organs of those to whom the gratification of the sexual passion is from the pressure of circumstances denied, predispose to neuralgia. But little experience in the treatment of the somewhat more matured women of the better educated classes is required to demonstrate the truth of this statement.

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[1] I must observe that there were scarcely any hysterical cases in my list.

The influence of *education* on the predisposition to neuropathic affections, especially at the age when through the development of the sexual organs a considerable impulse is given to the nervous system, has not been overlooked by Anstie. False or improper education in relation to religious subjects is especially to be dreaded, that kind of spiritual training in which the imagination is improperly directed, and the mind is constrained to occupy itself with the supernatural, as the best means of deadening and repressing the natural emotions of the healthy body. The result is but too often a morbid excitability and debility of the nervous system, which occasionally leads to neuralgia.

General disturbances of nutrition constitute a very frequent cause of neuralgia, since the trophic disturbance of the nervous system produces a strong predisposition to neuralgia. Amongst these anæmia and its various forms of oligæmia, hydræmia, and chlorosis may be specially mentioned; all anæmic conditions caused by loss of blood or other fluids, by bad digestion or by serious disease, and all cachectic states consequent upon severe diseases of any organ, malignant growths, etc., furnish a large contingent to the numbers of those predisposed to neuralgia. It may, in fact, be fairly maintained that anæmia in this wide sense of the term is one of the most important conditions in the etiology of this disease.

Associated with these are those general trophic disturbances which have been designated premature senility, including the changes of tissue that occur at a relatively early age, and which, as a rule, occur only at a more advanced period of life, and which manifest themselves in the form of various degenerative processes, such as induration, atrophy, fatty degeneration, calcification, and softening. These conditions are particularly common in cases of chronic alcoholism; they also occur in consequence of grief and anxiety, of overwork and dissolute habits, and constitute also the sequelæ of protracted and serious disease. The occurrence of atheroma in the arteries, of fatty degeneration of various tissues, the appearance of an arcus senilis in the cornea, the early turning gray of the hair, and curvature of the vertebral column, are amongst the most prominent signs of such trophic disturbances. Where these exist in a well-marked form, they indicate that trophic disturbances have commenced which pave the way to even the most severe forms of neuralgia.

The influence of *climate*, of *season*, and of *weather*, of *avocation* and *mode of life*, can only be so far regarded as predisposing conditions, as they may happen to lead to a greater or less co-operation of accidental causes of disease.

b. *Exciting Causes of Neuralgia.*

By far the most important of these are traumatic and mechanical agents acting upon sensory nerves. Many cases are recorded where, in consequence of punctures and incised wounds, of contusions, gunshot wounds, and other injuries of nerves, violent and widespread neuralgiæ have arisen; and in many instances this effect has been the result of injury done to some small and purely sensory branches, as, for example, in venesection, or in wounds of the nerves of the fingers, and the like. All these circumstances, which cannot be separately men-

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tioned, act, as it would appear, either by occasioning inflammatory changes (neuritis), or by forming tumors on the nerves (traumatic neuromata, amongst which the neuromata following amputation are the most frequent causes of severe neuralgia), or, lastly, purely mechanically, by pressure and laceration in consequence of the retention of foreign bodies in the wound. It must be remarked, however, that when such consequences of injuries occur, they by no means lead invariably to neuralgia, and that some further though still unknown change must take place in the nerves before neuralgia is established.

In a similar but insufficiently explained manner to these traumatic injuries, numerous and varied *pathologico-anatomical changes* in the nerves occasion neuralgia. On the one hand, acute or chronic neuritis, taking place in the neurilemma, is very frequently an accompaniment of the disease; whilst, on the other hand, it is often caused by new-formations on and in the nerves—that is, all that is included under the names of true and false neuromata (see the section on Neuroma), and the small, hard, movable tubercula dolorosa which have hitherto been insufficiently studied, the cancerous and cystiform new-formations on the nerves, and the like. Moreover, degenerative and softening processes taking place in the nerve trunks are occasionally found to be the causes of neuralgia.

Neuralgiæ depending upon purely mechanical conditions are especially induced by changes in the organs and tissues adjoining nerves, and in this category may be enumerated, as particularly frequent causes, diseases of the periosteum and of the bones, for in the close relations that the nerves bear in certain parts of the bones (as in the interior of canals, foramina, furrows, grooves, etc.), it is very natural that periostitis and ostitis, with their results, that caries and sclerosis of the bones, hyperostoses and exostoses, should be capable of mechanically irritating nerves in the most varying manner. There are certain nerves which have to traverse long and narrow bony canals, such as the various branches of the fifth, that are especially exposed to such irritation, and are thereby particularly predisposed to neuralgia. The view of Anstie, that the periostitic thickenings so frequently present must not always be regarded as the causes of neuralgia, but that they may themselves be the consequences of it and of the trophic disturbances produced by it, is here worthy of mention.

Just as in those cases where disease of the bone is present, all conceivable enlargements and tumors may cause neuralgia by pressure upon the nerves. *Aneurisms* frequently act in this way, but it is still doubtful whether *varicosities* and *dilatation* of the veins can produce the same effect. Venous stasis is indeed frequently spoken of as a cause of neuralgia, and when nerves and veins pass in company through narrow apertures and canals, it cannot well be denied that some mechanical irritation of the nerve may be produced by the pressure of the swollen vein ; but it is only necessary to refer to the comparative rarity of neuralgic affections in cardiac and pulmonary disease, in which there is a high degree of venous stasis, to show that this condition can only be effective where there is a strongly marked predisposition to neuralgic disease. *Herniæ* may occasion neuralgia by their mechanical action, as in cases of obturator and ischiatic ruptures. *Pregnancy*, as well as changes in the form and position of the uterus, and swellings of other organs, as of the liver, kidneys, ovaries, etc., may all become causes of neuralgia by pressure. The same may occur with fecal accumulations in the intestine, with concretions in various parts of the body, and lastly, with all sorts of new-growths, whatever may be their position, as soon as they begin to implicate the sensory nerves. Many hiatuses unfortunately exist in regard to the precise mode in which these mechanical conditions induce neuralgia ; but it is certain that they are not always effective, and it is probable that a very definite alteration in the nerves must occur before the symptoms we include under the term neuralgia supervene.

An injurious influence of great importance, though its action is very obscure, is cold. No doubt can exist in regard to the fact that a great number of neuralgiæ are induced by those injurious agencies which are ordinarily considered as causes of "catching cold," such as a draught of air, exposure to wet and cold, raw winds, sleeping on moist earth, etc. The influence of the seasons appears to be reducible to the varying intensity of such causes, neuralgia originating most frequently in the cold and moist winter months. Those nerves that, owing to their anatomical position, are most exposed to these influences, as, for example, the fifth and the sciatic nerve, are most commonly affected with this kind of "rheumatic" neuralgia. We are at present wholly unable to give a satisfactory account of the anatomical changes underlying the rheumatic form of neuralgia, though the opinion is gaining ground that slight inflammatory conditions of the neurilemma are commonly present (hyperæmia, swelling, fluid exudations, etc.). But in regard to the pathogenesis of these rheumatic disturbances of nutrition, and the mode in which exposure to cold brings about this disturbance, we are quite ignorant, and any digression upon the point would consequently be here out of place.

The action of definite poisons, such as those which produce various infectious diseases and poisoning (when absorbed into the blood) in the development of neuralgia is not less enigmatical, yet no doubt can exist that neuralgia, may be produced by these agents. Among the best known and most frequent causes of neuralgia are malarial infections. In malarial regions neuralgia is very common, and, like intermittent fever, occurs here and there sporadically. It is worthy of note that in such cases certain nerves, and especially the first branch of the fifth, are particularly liable to be affected. These malarial neuralgiæ have a well-marked periodic course, and are often accompanied by febrile phenomena, and yield very readily to anti-periodic means. Still, it can scarcely be maintained that every neuralgia yielding to quinine depends on malaria, since many others, and especially rheumatic affections, exhibit a distinctly marked periodic course; it is only in those cases where a tertian or quartan type is present that the malarial origin of the disease can be positively affirmed. The great majority of these malarial neuralgiæ, according to Griesinger,[1] occur in somewhat advanced age, above the age of forty. How far other infectious diseases can give occasion to neuralgia has not been satisfactorily determined. Nothnagel, however, mentions the occurrence of genuine neuralgia of the supraorbital and occipital nerves in the first stage of typhoid fever (clearly distinguishable from ordinary headache). On the other hand, neuralgia not unfrequently occurs as a sequela of typhoid and other infectious diseases, but then it depends upon anatomical changes in the nerves themselves. It is more than doubtful whether *dyscrasiæ* can be regarded as causes of neuralgia. In former times no one doubted the existence of "rheumatic," "arthritic," "scorbutic," and "scrofulous" neuralgiæ; but at present the pain experienced in constitutional rheumatism, gout, or other similar forms of disease, is, in part at least, referred to other than purely neuralgic conditions, and in part the causes of actual neuralgic pains are looked for in more secondary conditions, such as propagation of inflammatory processes from the joints to neighboring nerve trunks, deposition of calcareous salts in them, disease of the blood-vessels, etc. The action of *syphilis* in producing the disease is in many cases much more obvious; thus syphilitic osteal and periosteal diseases may affect the nerves, or specific syphilitic processes (gummata, chronic inflammatory processes in the connective tissue and in the blood-vessels, etc.), partly affecting the peripheric nerves, partly the central organs, may be the causes of neuralgia. The well-known *dolores osteocopi*, however, should not be mistaken for neuralgiæ. Fournier[1] has recently referred to true neuralgiæ which belong to the stage of secondary syphilis, and occur with tolerable frequency, particularly in women. These neuralgiæ affect especially the supraorbital and sciatic nerves.

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[1] Infectionskrankheiten, 2. Auflage, p. 48.

Of the several forms of toxic disease, the influence of *lead* is that which is most certainly known to produce neuralgic affections (lead colic, perhaps also lead arthralgia). *Copper* and *mercury* may also be mentioned. In strongly mercurialized individuals we very frequently see obstinate and not easily curable neuralgiæ develop, which affect the body more or less generally.

Alcohol and *tobacco* may be mentioned in this point of view as organic poisons, but the mode of action of all these poisons is still very obscure.

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[1] *Fournier*, Leçons sur la syphilis, 1873, p. 774.

Diseases of the central organs of the nervous system constitute one of the most important groups of causes of neuralgia. They act upon the central paths and apparatus for the conduction and reception of sensations in the spinal cord and brain, and in accordance with the laws of excentric projection the neuralgiæ thus produced are referred to the peripheral nerve paths. It is at the same time to be remarked, that much of the pain thus excited is not really neuralgic (and hence is best described as "neuralgiform"), and that true neuralgiæ occur only when the pathological conditions in question operate directly on the sensory central apparatus. The diseases which here deserve mention are hyperæmia, inflammations, softening of the brain and spinal cord, tumors of all kinds, scleroses, tabes dorsalis, etc. It is obviously a matter of the greatest importance that the physician should be able to recognize such processes as causes of neuralgia.

Lastly, a series of causes are mentioned in books, the mode of action of which is still extremely obscure, and it is even doubtful whether they can properly be regarded as real causes of the affection. Amongst these are *irritations of peripheric organs*, which are probably transferred in the central organ to remote sensory paths, in which they excite neuralgiæ. Amongst the most probable cases are those where injuries or disease of sensory nerves have ultimately led to neuralgiæ of more or less remote nerve paths. Thus Anstie mentions two cases of neuralgia of the fifth, one of which could with tolerable certainty be referred to an injury of the ulnar nerve, and the other to an injury of the occipital nerve. Analogies with the processes of irradiation of sensation here readily suggest themselves, as well as the idea of a propagated neuritis gradually developing itself in the centre. A strongly developed predisposition also materially favors the occurrence of such diseases. Closely allied to these cases are those neuralgiæ which not unfrequently result from *overwork of the eyes*, and chiefly affect the fifth nerve; and with these may be associated the neuralgiæ consequent on *caries of the teeth*. Neuralgiæ not unfrequently proceed from *irritation* and *diseases of the generative organs*, as venereal excesses, masturbation, blennorrhœa and epididymitis (Mauriac), diseases of the uterus and ovaries, etc., though the pain by no means always affects the nerves supplying the genital organs, but occasionally those of quite distant regions. The relations that exist between diseases of the intestines, intestinal worms, habitual constipation, hepatic obstruction, and the like, and neuralgiæ, are very obscure, and are for the most part only inferred from the results of therapeutic interference; still, there appear to be several well-authenticated cases on record in which such a connection existed.[1] Suppressed perspiration of the feet, and suppressed cutaneous secretion, etc., have formerly received far too much consideration as causes of neuralgia, though perhaps their influence cannot be entirely denied.

Pathological Anatomy and Pathogenesis.

We have now enumerated a great number of very different lesions as the causes of neuralgiæ. It is evident that an accurate knowledge of the pathologico-anatomical changes occurring in neuralgia can alone enable us to appreciate the finer points of connection between cause and effect, and consequently enable us to understand thoroughly the essential character of neuralgia. But if we examine more closely the facts above given, with reference to the anatomico-pathological changes, we shall soon discover our ignorance of the essential changes of the sensory nervous apparatus in neuralgia, and that these changes are inaccessible to the means of investigation we at present possess. We cannot possibly expect to meet with coarse anatomical changes; a nerve may at one moment be in a state of the most violent excitation, whilst the very next instant it may be performing its functions in a perfectly normal manner, and since a perfect intermission of the painful phenomena may thus occur, it cannot have undergone any notable anatomical changes; every such change would be accompanied by considerable interference with the function of the nerves, that is to say, with a high degree of anæsthesia. It is hence more than probable that the coarser anatomical changes so frequently found are not the essential feature of neuralgia, and Anstie is quite justified in stating that the anatomical changes are simply accidental, and are only rarely to be regarded as factors in the production of neuralgia.

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[1] See *Bretschneider*, loc. cit., and *Stromeyer*, Lokalneurosen, 1873.

The conditions that have been hitherto discovered may be given in a very few words. They consist in the flattening and atrophy of the nerves, a degeneration of the nerve fibres, swelling and hyperæmia, inflammation and thickening of the neurilemma, dilatation and tortuosity of both the arteries and veins of the nerve trunks, and cicatricial indurations and growths in them, and similar changes may be observed in the central nervous system. The great number and variety of the morbid changes that have been discovered is sufficient evidence that they are not essential to neuralgia, whilst in many cases the most careful investigation has failed to indicate any changes at all; at the same time it must be acknowledged that the negative result of such examination by no means proves that slighter or more transient morbid changes may not have existed during life (as, for example, hyperæmia, slight exudation, œdema, and the like), and in particular does not disprove the opinion expressed by several observers, that slight neuritis is at the bottom of all neuralgiæ. Upon the whole, additional information on this point can only be expected from researches undertaken, with all the more delicate means of research employed in modern microscopy, upon perfectly fresh specimens, which may best be obtained by resection.

From what has been stated it is obvious that the pathogeny of neuralgia is still extremely obscure, and for the most part rests on hypotheses. Thus in relation to the circumstances which cause a predisposition to neuralgia, the terms debility of the tissues, molecular disturbances of nutrition, greater vulnerability, greater excitability of the sensory nerves, are all expressions which but lightly veil our ignorance of the essential nature of the process. We shall hereafter perhaps obtain more definite information in regard to it.

As regards the exciting causes, it is admitted that in some instances they act as direct irritants to the sensory nerves, and thus excite neuralgia, as for instance in the case of wounds, foreign bodies, mechanical compression, and the pulsating pressure of aneurisms, etc. In other instances the same trophic disturbances which cause neuralgia—here usually called hyperæsthesia—may be induced by inflammatory changes, and there are still other conditions, as for instance anæmia, infection, and poisoning, which may directly produce hyperæsthesia, the slightest irritation being then capable of calling forth the neuralgic phenomena.

In opposition to this it may be stated positively that the pains produced by direct stimulation of the sensory nerves differ essentially from neuralgic pains, in that these last occur some time after the stimulus has been applied; that with continued application of the stimulus they can intermit, and that with cessation of the stimulus they can continue; that the pains experienced in neuralgia caused by aneurisms are not isochronous with the pulse. It may be remarked also that hyperæsthesia and neuralgia are by no means convertible terms, and that the neuralgic phenomena do not succeed each other in the same order as those of hyperæsthesia. The conclusion is therefore inevitable that *neuralgia is something essentially different from the sensory impressions caused by the immediate action of stimuli on the nerves*, and that thus the pain resulting from a wound or a foreign body represents something essentially different from the neuralgia induced by the same stimuli. It is easy to conceive that under the influence of definite causes a definite alteration is effected in the nerves which really represents neuralgia. As the cardinal symptoms of neuralgia are remarkably similar in all cases, the further conclusion may be drawn, that the *most diverse etiological influences always induce the same changes in the nerves*, and in this sense neuralgia, or at least by far the majority of cases of neuralgia, may very well be regarded as constituting a well-defined form of disease.

In what this peculiar condition of the nerves consists, which is capable of continuing after the cessation of the causes that have called it forth, and which can produce those peculiar phenomena we designate as a neuralgic paroxysm, we are absolutely ignorant. Our means of research are as yet incapable of investigating it more closely. Still this much may be said, that in *neu-*

ralgia we have to do with a quite definite and peculiar form of trophic disturbance in the sensory nervous apparatus.

In regard to the ordinary seat of this trophic disturbance, nothing accurate is known, but it is probable that the seat varies, and this much appears certain, that for the most part a definite group of fibres (or their central terminations), as they are combined to form a nerve trunk or branch, is affected. At what place in the length of the nerve this is present it is difficult to say, and perhaps it may be at any height. The peripheric fibrils may be affected at various points, and for various lengths of their course; or the posterior roots, and their prolongations in the spinal cord, may be the seat of the neuralgic trophic disturbance; or lastly, the central fibrils running in the spinal cord and brain may be affected up to the terminal central apparatus. The investigations that have hitherto been made have acquainted us with many important facts, but have furnished no very satisfactory conclusions.

The present state of our knowledge does not permit us to go beyond the statements that have here been made. It would appear therefore that all views that have been definitely advanced in regard to the essence of neuralgia, as well as every scientific theory respecting it, are premature.

This is not the place to subject the various views and theories on the essence of neuralgia to a profound criticism. The greater number of authors avoid expressing any definite statement in regard to this point; their definitions of neuralgia are simply short accounts of its chief symptoms. The only one that here deserves mention is a highly suggestive one that has recently been propounded by Anstie, who has endeavored to support it with much ingenuity. Anstie believes that all true neuralgiæ have their seat in the posterior roots of those spinal nerves in which pain is experienced, and that the specific change consists in atrophy of the posterior roots, and of the adjoining central fibres and ganglion cells, though he does not consider that this is commonly a result of inflammation. We must admit that the very full grounds for this opinion advanced by Anstie have not convinced us of its accuracy. There is no positive evidence in its favor, and we can only allow that it possesses a certain amount of probability for many forms of neuralgia, and especially for the constitutional, the central, and so-called reflex neuralgiæ. But we are of opinion that but few would with Anstie regard it as demonstrated that every neuralgia has its seat in the posterior roots of the spinal nerves, and still less that these are always affected with atrophy, or incipient atrophy. Quite independently of the absence of all necroscopic evidence, on which we lay but little stress, insuperable difficulties appear to be offered to this theory by the traumatic neuralgiæ (especially those arising from gunshot wounds), by the limitation of the disease to individual nerve trunks which receive their fibres from different roots, and finally by the ease with which many neuralgiæ can be cured, and the efficaciousness of many remedies which act upon the peripheral terminations. The view, in fact, requires a closer testing by careful anatomical investigations.

The proposition laid down by Benedict that all neuralgiæ, at least all peripheric neuralgiæ, are due to slight neuritis, can, in the present state of our knowledge, neither be proved nor disproved; it should, at all events, be considered true only of certain etiological forms of neuralgiæ. The insufficiency of the view of the cause of neuralgia, advanced in Niemeyer's well-known text-book, is sufficiently clear from what has been stated; a painful excitation of the sensory nerves does not constitute a neuralgia, because it is produced by unusual stimuli, or in some unusual place; there must always be a certain change in the nerves themselves.

Eulenburg[1] propounds a view in regard to the essential change taking place in neuralgia similar to the above, but acknowledges our profound ignorance of the kind and mode of operation.

SYMPTOMATOLOGY.

1. *General Characters of the Disease.*

For the most part the various forms of neuralgia are preceded for a longer or shorter time by a series of premonitory symptoms which are more or less strongly marked: slight twitchings, and now and again monitory pains are experienced in the nerve region that is about to be affected; pricking sensations are also felt, which recur more and more frequently; often formication also, or abnormal sensation of cold, and rarely a feeling of general indisposition. It is but seldom that neuralgia supervenes quite suddenly, and rises at once to its full intensity. The neuralgic attack or paroxysm is specially characterized by the occurrence of the most severe pain in a definite region of the body, or in a definite nerve territory. The pain may present different characters; for the most part the patient describes them as boring, tearing, cutting, darting like lightning, and more rarely as burning. Their intensity varies from moment to moment, and they may gradually rise to the greatest severity, when the sufferer can no longer find words to describe them, whilst he gives vent to the wildest exclamations, and is urged almost to desperation. If the nerve be examined during an attack, several very painful points may be found, which are extremely sensitive to pressure (painful spots, Schmerzpunkte, points douloureux), and appear to be the centres and starting-points of the pain. In many cases more or less distinctly marked hyperæsthesia, and in others anæsthesia of the nerve region affected is present.

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[1] Loc. cit., p. 51.

At the height of the paroxysm irradiation of the pain frequently occurs to more or less remote sensory nerves, and especially to the symmetrical nerves of the opposite side of the body. In neuralgiæ affecting mixed as well as purely sensory nerves, distinct motor phenomena are not unfrequently seen, such as trembling, tonic and clonic cramps, etc. The mobility of the part is almost always interfered with, on account of the intense pain that accompanies every movement, and, more rarely and at a later period, in consequence of actual motor debility or paralysis.

Vaso-motor phenomena occur early in the disease, and are often very remarkable; many attacks commence with singular paleness of the skin, which is soon exchanged for increased redness. Horripilation, as well as persistent and abnormal sensations of cold or heat, consequent on anomalies of the cutaneous circulation, are not unfrequent.

Disturbances of secretion also occur, especially in neuralgiæ affecting nerves that ordinarily govern the functional activity of secreting organs. Copious lachrymation and profuse salivation are amongst the most frequent of such phenomena; increased urinary secretion and sweating are also occasionally met with.

General indisposition is scarcely ever an accompaniment of the attacks; the thermometer shows no increase in the temperature of the body; the pulse remains of normal frequency, or is somewhat retarded; the psychical functions remain intact, or at most, when the paroxysms are very violent, become somewhat impaired by the intensity of the pain.

A neuralgic attack presenting such symptoms as these may last for a very variable period, sometimes ceasing in the course of a few minutes, at others continuing for half an hour, or for several hours, during the whole of which time the patient is racked with pain. The attacks seldom last longer than this, or if they do, they may be regarded as a series of attacks following one another with rapidity. As the attacks draw to an end, the pain sometimes ceases quite suddenly, but for the most part it gradually diminishes, the commencing intermission being often interrupted by a few lightning-like pains. A condition of complete or relative freedom from pain, with a corresponding feeling of comfort, now sets in, and it is only after very violent attacks that any exhaustion or depression remains.

A period now follows in which pain and all the other phenomena of the disease entirely disappear;—we have then complete intermission of pain; or some slight pain and tenderness remain, and the painful spots are still present;—the neuralgia is then said to have remitted. This period may also last for a very variable period, on the duration of which the return of the attack depends, and this may occur in the course of a few minutes, or of some hours, days, weeks, or even months. The return of the disease after an interval of a certain number of hours or days is not uncommon; the affection is then said to be a typical or periodic neuralgia.

The recurrence of the attack often takes place without any appreciable cause, that is to say, quite spontaneously; there are certain conditions, however, which are in many cases capable of immediately inducing an attack, such as slight irritation of the skin, a draught of cold air, movement of the affected part, the performance of certain natural functions, psychical emotions, etc., and the patient having acquired a knowledge of these by experience may be observed to avoid them with the greatest solicitude.

As the disease progresses, the severity of the attacks increases to a certain point, at which it remains stationary or commences gradually to decline. The attacks often recur with special frequency at certain times and occasions. In old and protracted cases other phenomena make their appearance, such as trophic and severe motor disturbances, disorder of the general health, anæmia, extreme nervousness, and finally even psychical ailments may complicate the picture of the chronic form of neuralgia.

The number and violence of the attacks undergoing gradual diminution, more or less rapid recovery takes place, which is the most common termination of the disease; but instances are not wanting in which it may become chronic, and persist for years; and lastly, it may be quite incurable, and the patient once afflicted with it may remain so for the rest of his life.

The above general account of the disease, though drawn from the observation of a great number of cases, is by no means exhaustive, and, on the other hand, as the consideration of the several symptoms and the different forms of neuralgia will show, all these phenomena do not appear in every case.

2. *Analysis of the Several Symptoms.*

Pain.—This is decidedly the most prominent of all the phenomena in every form of neuralgia. It does not, however, present any such perfectly characteristic feature as will enable us at once to recognize it as neuralgic. It is a purely subjective symptom; we can only acquire a knowledge of it from the statements of the patient, and these statements differ materially in different cases. The pain is described as tearing, boring, stabbing, dragging, and sometimes as tensive or lightning-like, sometimes as glowing or burning,[1] or as if proceeding from the cut of a knife, and it may be felt either as if it were on the surface or deeply seated and in the bones; descriptions of pain are sometimes given to the effect that it is like what might be caused by drawing the nerve through a narrow ring, or as if the part of the body were being torn asunder, etc. In one case the pain will be felt shooting from the centre to the periphery, in another in the opposite direction (descending and ascending

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[1] *Weir Mitchell* (Injuries of the Nerves, etc. Philadelphia, 1872) describes, under the name of causalgia, a very frequent and characteristic form of pain following gunshot wounds, which consists of a violent burning pain occurring in the peripheric region of distribution of the nerve affected, and especially in the hand and foot, and which can only be assuaged by the constant application of cold water to the skin.

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neuralgia); in short, very different accounts are given, which, however, have this feature in common, that they describe the *pain* as being of *great severity*, and especially of very *varying intensity.* The localization of the pain likewise varies very much; sometimes it is in a fixed spot, sometimes it changes about among several; usually it is felt at some depth beneath the skin, and it is often directly referred to a nerve trunk, so that patients who are perfectly uneducated will frequently trace the course of a nerve, with great precision, in their description of the painful parts. This fact, that the trunk of a nerve appears to be painful throughout a certain part of its course, is most easily explained by conceiving that the nervi nervorum, so minutely described of late by Sappey and others, are also implicated.

The pain of neuralgia is capable of being augmented in intensity by various external conditions: amongst them we may particularly notice motion of the affected part, which is much dreaded by the patient; so that in facial neuralgia, speaking and mastication,—in intercostal neuralgia, deep respiration—and in sciatica, the movements of locomotion are carefully avoided. In some rare instances the pain is relieved by movement of the part. Irritation of the skin, cold air, cold water, and especially the light contact of external bodies with the skin, produce an attack of pain; whilst it may often be seen that the patients are relieved by firm pressure on the painful part, and frequently resort to the strangest means of obtaining this relief. There is great difference in the intensity of the neuralgiæ of the different nerves; it is true that all the forms present cases of very great, and of less intensity, nevertheless it appears, from accounts given by patients, that the most atrocious pain is felt in severe cases of neuralgia of the fifth nerve, and next to this in point of severity is the pain in sciatica. Attempts have been made to draw some inference as to the localization of the cause of the pain from its varying intensity and quality (Benedict). But this does not appear to afford much help to us, and at most it may only lead us to diagnosticate, with some degree of probability, neuralgic pains brought on by some peripheric cause, from those that are produced by a central cause (excentric). (See for further details the section on Diagnosis.)

Moreover, it would appear from a general consideration of neuralgic pain, and from the mode in which it spreads, that we have not so much to do with excitation of the peripheric terminations of the sensory nerves, as with excitation at certain points in their course where numerous sensory fibres unite to form fasciculi and trunks, whether this be in peripheric nerves, or in the posterior roots, or in the central organs.

Painful Points.—These, which were originally alluded to by Bérard, were first carefully investigated and described by Valleix, under the term "*points douloureux*," and have been the object of numerous researches and much discussion. Their significance in the pathology of neuralgia has been much overestimated. The facts are simply these: If we examine the part which is the seat of pain during a neuralgic attack, we shall usually find one or more points that are extremely sensitive to the pressure of the tip of the finger or any similar body. These points are usually quite circumscribed, and sometimes are, and sometimes are not, the seats and centres of radiation of spontaneous pain. Pressure upon them produces very acute pain, and is capable of increasing the intensity of the neuralgic attack, or even of bringing one on. These painful points are most strongly marked during the paroxysm, and their sensitiveness stands in nearly direct relation with the intensity of the pain; in accordance with this we find that they are either altogether absent or but slightly painful in the period when the pain has remitted, though occasionally cases occur in which the painful points are discoverable in the periods of remission, and in some instances pressure on them will produce an attack. Further inquiry shows that in order to excite pain the pressure must be directed upon the parts lying at some depth beneath the skin, since, if a fold of the skin is pinched, a different kind of pain is felt than that which is experienced when the painful spots are pressed. Care should therefore be taken not to confound the coincident hyperæsthesia of the skin over the painful points with the pain caused by pressure upon them. If the pressure is increased to a certain degree, and then maintained for some time, the pains often disappear gradually, and a certain period must then elapse before the same point again becomes painful on pressure.

Careful investigation shows clearly that all or the greater number of painful points are to be found in those parts of the skin beneath which nerve trunks or branches lie. As a general rule, there are only a few points in the course of a nerve which are thus painful, but it is not uncommon to find the nerve sensitive to pressure throughout its whole course. There are certain parts of the nerve that are specially liable to present such painful points, such as the points of emergence from apertures, canals, or grooves in bone, the points where aponeuroses or muscles are traversed by nerves, or where these divide or give off branches, and where they lie on a hard bed and can be easily compressed. It is obvious, then, that in the greater number of neuralgiæ several painful points may be found, and that particular nerves present anatomical peculiarities that are favorable to the occurrence of such points. Valleix has studied and established the position of painful points with great care, and we shall describe them in detail when speaking of the several forms of neuralgia. Trousseau states that in all neuralgiæ the spinous processes of the vertebræ beneath which the nerves affected are given off from the cord are painful on pressure, and regards this painful point, which he terms the "apophysial point," as a constant, and, in a diagnostic point of view, extremely important criterion in neuralgia.

As a rule, no changes are observed in the painful points; the skin is neither reddened nor swollen, nor is anything abnormal to be found beneath them, though Anstie states that in some cases the periosteum has been found to be somewhat thickened. The same observer also states that the painful points are in many cases absent in the beginning of the neuralgia, and only make their appearance after it has lasted some time. The discovery of the painful points is effected by careful palpation with the fingers, and the pathological conditions present may be recognized with great facility by a comparison with the corresponding part of the opposite side.

The frequency of the occurrence of the painful points in neuralgia has also been considerably overestimated by Valleix, who, in fact, appears to have never failed in discovering them. Nearly all the recent and more careful observers are, however, opposed to him on this point, as Romberg, Schuh, Hasse, Eulenburg, Trousseau, Anstie, etc. Romberg has drawn attention to certain conditions which perhaps afford an explanation of the differing statements made by different observers, as, for example, the varying amount and duration of the pressure applied, the sensitiveness of the nerve trunk and of the superjacent skin. Eulenburg (loc. cit., p. 43) states that he found them in rather more than half the cases that came under his care, whilst he failed to discover them in the remainder. I may also remark that in a considerable number of my own cases it was impossible even with the most conscientious care to demonstrate their existence. But if this be the case, the semiological and diagnostic value attributed to them by Valleix is considerably diminished, and no conclusion can be drawn from their absence in regard to the diagnosis of neuralgia in any particular case.

The pathological significance of the painful points, and their pathogenesis, are undoubtedly obscure, and very different opinions have been expressed in regard to them. It has been suggested, for example, that some local irritation or inflammation is always present in the nerve, and Lender has endeavored to establish this view by contending that such local inflammation constitutes the primary and essential feature of neuralgia. Anstie considers it to be probable that the painful points are in most instances the result of vaso-motor paralysis, the mode of action of which, however, he does not further explain. Eulenburg draws attention to the fact that where local centres of irritation exist, the irritations proceeding from them peripherically may occasion abnormally strong excitations—the so-called conduction hyperæsthesiæ—and that some of the painful points may perhaps be referable to these. It may further be noticed that in general hyperæsthesia of the entire length of the nerve, certain points of it, owing to their anatomical disposition, will be specially exposed to pressure, and, consequently, liable to the formation of painful points. Lastly, the participation of the nervi nervorum in the hyperæsthesia may not be uninfluential in producing great local sensitiveness to pain. No positive evidence, however, can be adduced for any of the foregoing possible causes of painful points. And it is probable that in different instances one or the other may be the real one. We must rest contented with having established the fact that in the course of neuralgic affections of sensory nerves, conditions of increased excitability, which are for the most part of a transitory nature, occur at certain points, and render them painful on pressure. It is, consequently, not improbable that this exalted excitability, like the neuralgic pain itself, is only a symptom (Theilerscheinung) of that still imperfectly understood trophic disturbance in the nerves which we have already sought to render probable as the essential cause of neuralgia; from the various seat and various extent of this trophic disturbance the presence or absence of painful points in the several cases of neuralgia would then receive its explanation.

Concomitant Sensory Symptoms.—One of the most frequent phenomena in neuralgia is the *irradiation of the pain* to other sensory nerves. When the paroxysm is at its height an extension of the pain to neighboring, and not infrequently to more or less distant nerves, is usually observed to take place in the first instance to other branches of the same nerve trunk, as, for example, when one branch of the fifth is primarily affected it spreads to the two others; next, to the corresponding nerves on the opposite side of the body, as, for example, from one sciatic to the other; and lastly to quite different nerve territories, as from one of the intercostal nerves to the fifth. These radiated pains are in general less intense than the original one, their duration is shorter, they begin to be felt when the paroxysm has reached its height, and disappear again before it has completely subsided. They are particularly liable to occur in nervous and excitable subjects. We can only explain this by supposing that in the gray substance of the central organs the stimulation of sensory ganglion cells extends, in consequence of their manifold intercommunications, to other ganglion cells not primarily affected, and is then referred to the periphery in accordance with the law of excentric projection. As in such transference of excitation to nerve paths not usually or physiologically in action, great resistances have to be overcome, it is evident why the radiated pains first occur during the height of the paroxysm, and for the most part in those who are highly excitable. We have no more precise information on this point. The cases of spreading and transference of neuralgia from one nerve territory to another are to be distinguished from these simply irradiated pains, though perhaps the conducting paths are the same in both instances. It is not uncommon for neuralgia of one branch of the fifth nerve to shift to another, or for neuralgia to pass from the occipital to the fifth nerve, in which case there is a certain amount of independence in the affection. In such cases we are dealing with an actual extension of the neuralgic trophic disturbance, which probably takes place by the intermediation of the central organs; in irradiation, on the other hand, we have a transient excitation which is simply transmitted from another centre of irritation, and is dependent upon this. The alternation and metastasis of neuralgiæ from one nerve territory to another cannot be regarded as a phenomenon of irradiation, but rather as a local change of place of the neuralgic trophic disturbance, which is doubtless also effected through the central nervous system.

In addition to the neuralgic pain there may be observed. though not very frequently, in the region to which the affected nerve is distributed, subjective or various paræsthesic sensations of a peculiar kind, which are variously described as formication, creeping and woolly sensations, and numbness. These sensations frequently persist through the period of remission, but are less distinctly perceived, or at least are less regarded during the paroxysm, on account of the attention being directed to the pain. Their nature and mode of origin still remain obscure, though amongst the more remote causes macroscopic lesions of the affected nerve may be mentioned, and especially is it the case that persistent and strongly marked sensations of numbness are suggestive of the development of organic disease in the nerve trunk. The slighter grades may, however, on the other hand, be produced by other causes.

These subjective paræsthesiæ are not unfrequently associated with disturbances of sensibility which can be proved objectively, and which, moreover, often exist without subjective sensations. These disturbances show themselves as *hyperæsthesia or anæsthesia of the skin* in the region supplied by the affected nerves. Türck first described these phenomena in 1850, and ascertained that there was slight anæsthesia of the surface of the skin with coincident hyperæsthesia of the deeper layers of tissue. Trousseau describes hyperæsthesia of the skin as a common symptom in many forms of neuralgia ; he also mentions anæsthesia, which always supervenes later than hyperæsthesia, and in all probability depends on deeper-seated changes of the tissues. Traube maintains that these symptoms may also be observed in those periods of the disease that are free from pain. Anstie found that diminution of the tactile sensibility was an *almost* constant phenomenon of neuralgia. Nothnagel has investigated these symptoms more minutely still, and has established the following facts in a great number of cases : In neuralgiæ of the nerves of the extremities, without any demonstrable anatomical lesions, an alteration of the tactile sensibility of the skin is invariably present, either in the form of hyperæsthesia (or, more correctly speaking, hyperalgesia) or of anæsthesia. These phenomena stand in a certain definite relation to the persistence and duration of the neuralgia. At the commencement hyperalgesia of the skin is present, and at a later period diminution of the sensibility. These disturbances, however, can for the most part only be discovered by very careful testing and comparison with the opposite side, because they are not strongly marked. Such symptoms are usually limited to the region of distribution of the affected nerve, but cases are also met with in which, with quite limited neuralgia, the concomitant disturbance of sensibility affects the whole of the same side of the body. As a rule (though certainly not without exception) in recent neuralgiæ (having a duration of from two to eight weeks) there is hyperalgesia of the skin; in older neuralgiæ, anæsthesia. The modifications of sensibility are also present, though less strongly marked, in the painless intervals; after the disappearance of the neuralgia they usually quickly vanish.

I have paid particular attention to these symptoms in a large number of cases, and have satisfied myself that Nothnagel's statements are in general correct. I have, however, met with a few cases of pure neuralgia, in which neither hyperalgesia nor anæsthesia could be detected by the most careful investigation. In several instances, moreover, I have observed well-marked anæsthesia at a very early period (within the first week); whether the diminution of sensibility affecting one-half of the body, observed by Berger[1] after gunshot wounds (without lesion of any large nerve trunk), belongs to this category of symptoms or not may still be regarded as doubtful.

The explanation of these phenomena is beset with no small difficulties; the anæsthesia in the various forms of neuralgiæ is often referable to perfectly obvious causes; thus in many cases, in which anæsthesia of the conducting organs is present, the neuralgia may be attributed to the presence of tumors or other conditions producing pressure, to organic lesions in the central organs, or to peripheric neuritis; and in other cases the cutaneous anæsthesia may result from spasmodic contraction of the arteries. A number of cases, however, present themselves in which such conditions can neither be demonstrated nor even admitted to be present. Since Nothnagel has pointed out that similar disturbances of cutaneous sensibility occur in a variety of other painful (but not neuralgic) affections, it cannot be admitted that we are here dealing with a symptom of true neuralgic trophic disturbance, and the view of Nothnagel becomes not improbable, *that the pain itself occasions the variations of the cutaneous sensibility, including both the hyperæsthesia and the anæsthesia.* This, however, by no means clears up the matter, and further hypotheses are needed to explain the process. Nothnagel locates these processes, in most cases, in the central organ, and considers the anæsthesia to result from exhaustion produced by the pain, and the hyperæsthesia to be due to diminished resistance to conductivity on the part of the sensory central apparatus. This does not exclude the fact that increased or diminished resistance to conduction (conduction anæsthesia and hyperæsthesia) may be caused in the peripheral nerves by the neuralgia itself, which can, however, only afford an explanation of those disturbances of sensibility that affect the region of distribution of the neuralgically affected nerves.

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[1] *Berger*, Berlin. klinische Wochenschrift, 1871, No. 20.

(For further particulars in regard to this hypothetical attempt to explain the phenomena, see Nothnagel, loc. cit.)

Concomitant Motor Phenomena.—These are phenomena of extremely common occurrence in neuralgia, and two classes may be distinguished: 1, Those which result from the *direct* influence of the neuralgia, or of the morbid changes causing the neuralgia, upon the motor nerves; and 2, those that originate in an *indirect* manner, through the intermediation of the central nervous system, and therefore by reflex action. The former can, of course, only occur in mixed nerves or in various diseases of the central organs which affect both sensory and motor apparatus; the latter only in neuralgia of purely sensory nerves.

Direct motor disturbances appear either in the form of convulsive or of paralytic symptoms. All possible grades of motor irritation may be observed, from fibrillar contractions and slight muscular twitches to tremors, contractions, spasms, and even fully developed and severe convulsions, such results being not uncommon in sciatica. Distinct paretic or even paralytic phenomena are usually observed in the later stages of the disease, that is, muscular debility, gradually increasing to complete paralysis; and great care must be taken to distinguish these from that voluntary rest and immobility of the limbs which is occasioned by fear of bringing on an attack of pain. These symptoms are always limited to the region of distribution of the mixed nerves affected with the neuralgia. The explanation of these phenomena is to be sought, in the greater number of cases, in the gradual extension of the morbid changes causing the neuralgia, such as tumors, neuritis, contraction of cicatrices, to the motor nerves running in the same trunk; the incipient symptoms are consequently those of irritation, and the later ones, when the lesions are more serious, those of paralysis, the latter resulting from the more or less complete abolition of the power of conduction. It is quite intelligible that distinct paralytic symptoms may occur in motor nerves in cases where the sensibility has not been materially interfered with, since experience has shown that motor nerves are much less capable of resisting injurious agencies than sensory ones.

The reflex motor phenomena are exclusively those of irritation ; up to the present, at least, there has been no ground for the belief that reflex paralyses occur in the so-called external neuralgiæ, though from the results of Lewisson's experiments with severe irritation of the internal organs, there seems to be a possibility of their occurrence. The reflex phenomena of irritation are best marked and most frequently seen in neuralgiæ of the fifth nerve, where they appear in the form both of tonic and clonic spasms (blepharospasm and the like) in the muscles supplied by the facial; these symptoms must, however, be carefully distinguished from the direct motor disturbances occurring in the muscles of mastication, which are supplied by the fifth itself. In neuralgiæ affecting mixed nerves, phenomena of muscular contraction in the form of spasms, tremors, convulsions, contractions, etc., are not unfrequent. These can be the more certainly regarded as true reflex phenomena when the region affected is different from that supplied by the nerves affected with neuralgia. The explanation of all these symptoms may easily be deduced from the well-known physiological laws of reflex action. They are more violent, and embrace a wider area in proportion as the neuralgia is more intense, and in many light cases they are entirely absent. Their occurrence is favored by the nervous diathesis, and the condition that has been termed convulsibility. We may associate with these phenomena the fact observed by Türck, that the pulse diminishes in frequency in severe neuralgic paroxysms. Anstie, who corroborates this fact, even speaks of a transient but complete arrest of the heart's action. The latter observer also alleges that paralytic affections of internal organs containing smooth muscular fibres are amongst the most frequent phenomena in neuralgia, and mentions paralysis of the bladder, paralytic distention of the colon, etc., occurring as consequences of various neuralgiæ of the pelvic organs and of the external genitals. This, however, in my opinion, requires confirmation.

Vaso-motor Concomitant Symptoms.—The occurrence of these complicates, in many cases, the picture of neuralgia. Here, also, symptoms of irritation and of paralysis may be observed. At the commencement of the paroxysms contraction of the muscular tissue of the vessels is usually most marked, and the skin is, accordingly, then observed to be pale, and there is a sensation of coldness, etc., and these conditions may last a long time; but in most instances the phenomena of vaso-motor paralysis soon supervene, a diffused and more or less vivid redness of the skin, and of such parts of the mucous membranes as are visible, occurs, and some tumefaction, as well as those secretory and trophic disturbances which it is legitimate to refer, in part at least, to vaso-motor paralysis, may be present. Such vaso-motor disturbances may be particularly observed affecting the skin of the face, the conjunctiva, and the mucous membrane of the nose in facial neuralgia. Amongst the remaining neuralgiæ sciatica is that which is especially liable to be complicated with phenomena attributable to vaso-motor disturbance. These may either be limited to the region of the neuralgically affected nerves (that is to say, the territory of distribution of the vaso-motor nerves contained in them), or they are extended over a larger area, in which case conclusions can often be drawn in regard to the seat of the neuralgia. The larger vessels not unfrequently take part in these disturbances, the arteries appearing dilated and beating more strongly; whilst sphygmographic tracings supply evidence that at the commencement of the attack the tension of the arteries is increased, and that at a later period there is diminished tension and marked dicrotism of the pulse wave.

In vaso-motor disturbances, as in motor disturbances generally, a double mode of origin is possible, first, by direct, and secondly, by reflex action on the vascular nerves. Unfortunately the results of the numerous physiological investigations into the effect of reflex action upon the vaso-motor nerves are not sufficiently concordant to permit any positive conclusions to be drawn in regard to the behavior of the vaso-motor nerves in neuralgia; certain experiments have, however, demonstrated that reflex paralysis of vaso-motor nerves may be produced by powerful irritation of sensory nerves. We may with the more probability admit such paralysis of the vascular nerves, reddening of the skin, etc., to be reflex, when they prove to be widely distributed and not limited to the particular region supplied by the affected nerves. On the other hand, it must be remembered that such paralyses, like motor paralyses, may originate by direct action. The tetanic condition of the vaso-motor nerves may thus originate in a direct or in an indirect manner. As long as we are not in a position to distinguish the causes acting in a direct from those acting in a reflex mode, with certainty, and especially so long as we do not possess precise anatomical information in regard to the course, origin, and distribution of the vaso-motor nerves, and their relations to the mixed nerves running with them, it will continue to be futile to draw any definite conclusion from the presence and characters of vaso-motor phenomena in regard to the anatomical seat of neuralgia. It is only in the rarest cases that the diagnosis of the disease is materially facilitated and assisted by this means, and then only by the consideration of the accompanying symptoms, both motor and sensory, that may be present.

Concomitant Secretory Phenomena.—It may easily be conceived that the strong irritation of the sensory nerves that exists in neuralgia exerts an influence upon the secretions dependent upon these nerves. A considerable augmentation of the various secretions is often observed in neuralgia; thus we meet with abundant lachrymation, profuse nasal catarrh, and salivation in the several forms of neuralgia of the fifth, and augmented secretion of milk in neuralgia of the breast, etc. The excitation may, in these instances, be either direct or reflex, the latter being the most frequent. In many instances neuralgia affects the urinary and cutaneous secretions. In some instances the urine is abundant and watery, in others it is scanty. The secretion of sweat is often increased during the paroxysms, and they frequently terminate with copious perspiration. Nothing positive, however, can be stated in regard to the exact connection of these phenomena with the neuralgia.

Concomitant Trophic Phenomena.—Under this heading are included a whole series of various disturbances, including those which are usually held to be of an inflammatory nature, though after the insight that has recently been gained in regard to the essential changes taking place in inflammation these can no longer be regarded, in the ordinary sense of the word, as trophic disturbances. Two large groups of these so-called trophic disturbances may readily be distinguished from each other.

a. *Simple Qualitative and Quantitative Changes in the Nutrition of the Tissues.*—Amongst these the first to be mentioned is a *change in the color of the hair*, which has been repeatedly observed. The hair may become gray or white in the part to which the affected nerve is distributed, and according to Anstie it is not very uncommon for the discoloration to occur with each attack, whilst the natural color reappears after it; so again *an increase in the number and thickness of the hairs* has been occasionally observed by Romberg, Notta, and Anstie, an abundant growth of hair appearing in parts that were previously quite bare; lastly, *the hair may fall off more or less completely* in the neuralgically affected region. Anstie has also called attention to the greater roughness and darker pigmentation of the skin in those parts which are the seat of neuralgia, and analogous conditions are found in the *excessive formation of epithelium on mucous membranes;* for example, thicker epithelial coating on the tongue at the side corresponding to that of the face affected with facial neuralgia.

In other cases we meet with *hypertrophy and thickening of various tissues.* Thus, though very rarely, thickening of the periosteum and of the bones, increase of the subcutaneous layer of connective tissue and fat at the affected point, and even well-marked muscular hypertrophy occur (Grogan). Cases of atrophy and emaciation of tissue are much more common; thus we see attenuation of the skin, and disappearance of the subcutaneous layer of connective tissue and fat in facial neuralgia, whilst a certain amount of atrophy of the muscles is an ordinary phenomenon in sciatica and other neuralgiæ affecting mixed nerves; high degrees of muscular atrophy, for the most part, only occur in severe lesions of mixed peripheric nerve trunks, such as are produced by gunshot wounds, which have neuralgia as a consequence on the one hand, and paralysis on the other.

b. *Complex, generally Inflammatory Changes.*—These take place for the most part in the skin, or are here, at least, most familiar to us. Some few take place in the eye. Of those that occur in the skin the first that may be mentioned is simple erythema, which is closely allied to the redness caused by the vaso-motor paralysis. Then we have *erysipelas*—a true erysipelatous inflammation, presenting all the essential characters of this disease, limited to the region of distribution of the neuralgically affected nerve, and for the most part connected with very violent paroxysms; it is met with only in the face. Upon this point I am able to thoroughly corroborate the statements made by Anstie and others. The occurrence of *urticaria*, *pemphigus*, and similar affections, indicates a severe form of the disease, and the same may be said of those cases in which the skin is thin and red, as in "glossy fingers," and in those cases where the nails become deformed or fall off, and where intractable ulcers form on the skin. These are for the most part cases of symptomatic neuralgia, caused by severe wounds of peripheral nerve trunks, in which the neuralgia constitutes only a symptom, and often only a subordinate symptom of the whole disease, and does not stand in any intimate relation with the trophic disturbances. The same applies to cases of *herpes zoster*, which not unfrequently occurs in connection with well-marked neuralgia. This affection consists of a characteristic herpetic eruption (groups of vesicles appearing upon an inflamed, swollen, and reddened base) corresponding exactly to the region of distribution of a definite nerve or nerves. The herpes most commonly corresponds to an intercostal nerve (hence its zone-like form), and is next most frequently met with in the face, though it may occur in any nerve region of the body or extremities. The vesicles develop, in the course of a few days, on the originally reddened surface of the skin; they contain at first a little watery fluid, which gradually becomes cloudy, and dries up in the course of a few days to scabs and crusts, which after a while fall off, though they frequently leave behind them ulcers that heal with difficulty. The relation of herpes zoster to neuralgia is very inconstant; the proportion of cases in which it occurs is however very small, occurring only three times in 146 cases of neuralgia under my care, whilst, on the other hand, many cases of zoster are unaccompanied by neuralgia. This is particularly observable in young persons. Pain is often only present during the first few days, or it appears at the end of the eruption, and in many instances may remain in the form of severe neuralgia for a variable period after the disappearance of the eruption. In all the cases that have hitherto been subjected to anatomical investigation, as in those recorded by Baerensprung, Esmarch, Danielssen, Charcot and Cotard, Bahrdt, Wiedner, and Wyss, well-marked signs of inflammation, as redness, swelling, thickening, and serous and purulent infiltration of the neurilemma, have been discovered in the nerves or in the spinal ganglia, the region of distribution of which corresponds with that of the zoster. In complete accordance with these is the fact pointed out by Trousseau, and recently substantiated by McCrea,[1] that in herpes zoster evidence may be obtained of the nerves being seriously affected; in the first place cutaneous hyperæsthesia, and subsequently anæmia, discoloration, and well-marked anæsthesia. The latter symptoms have also been observed by Horner and Wyss. From all this it appears that zoster is a symptom standing in the most intimate connection with nervous disorders, though it is not an essential or characteristic feature of neuralgia. In those cases where it accompanies neuralgia, the conclusion may with some degree of certainty be drawn that the neuralgia is dependent upon neuritis.

Amongst the inflammatory disturbances affecting the eye, herpes is the one which most frequently results from neuralgia of the trigeminus. *Ophthalmia neuroparalytica*, with its deleterious consequences on the eyeball, is much more rarely observed. This is usually the result of more serious lesions of the fifth nerve, leading to anæsthesia. *Iritis* and *glaucoma* not unfrequently supervene upon neuralgia, as is shown by the observations of Anstie, Hutchinson, Wegner, and others, and by the physiological researches that have been made on the influence of the trigeminus upon the intraocular pressure. We shall return to this subject in speaking of neuralgia of the fifth pair of nerves.

The significance of these various disturbances of nutrition is at present very obscure. It appears at first sight most reasonable to attribute them to certain "trophic" nerves, and to refer

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[1] British Medical Journal, 1873, No. 647.

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all the above symptoms to the participation of these nerves in the disease. This is not indeed the place to enter fully into the difficult question of the existence of trophic nerves; yet there is no reason why we should not state that our opinion is entirely in favor of their existence, and that a whole series of the above-mentioned trophic disturbances are only explicable on the supposition that the nerves exert a direct influence upon the nutrition of the tissues, as, for example, the changes in the color and growth of the hair, the hyperplasia of the epithelium, the deposit of pigment, the hypertrophy and in part the atrophy of the tissues, and perhaps also a part of the inflammatory disturbances in the skin, erysipelas, pemphigus, herpes, etc. Admitting their existence, the view propounded by Charcot,[1] that trophic disturbances are a consequence of the excitation of these trophic nerves, would then be in every point of view probable. This view, however, which Weir Mitchell seems inclined to adopt, is not at present sufficiently well grounded. Other conditions, and especially vaso-motor conditions possibly present, may occasion at least a part of the disturbances observed. We are at present unable, even after Cohnheim's most recent investigations, to draw a sharp line between simple paralysis of the vessels and those changes of the vascular walls which are essential and determining causes of the inflammation, and it is conceivable that under certain circumstances, in those who are especially disposed to it, the nutrition changes necessary to the production of erysipelas, herpes, and the like, commence in the vessels by some action exerted upon the vaso-motor nerves. And there is still another possibility, which has been maintained and strongly supported by Friedreich,[2] that, especially in zoster, and in many forms of muscular atrophy occurring in the course of neuralgic affections, there is really present a gradually progressing neuritic process, which, starting from the primary focus of irritation to which the neuralgia is itself due, extends along the nerve to its finest ramifications, and ultimately reaches the skin, where it produces the characteristic inflammatory eruptions. However plausible this view is rendered by the arguments and observations of Friedreich, it is still deficient in a solid basis of anatomical investigation and experiment, and further, it only explains a part of the phenomena. Some, like Nothnagel, have referred the simple atrophy of muscles, that so frequently accompanies sciatica, to pure vaso-motor influences, and hold that the vaso-motor cramp present in these cases of neuralgia furnishes an adequate explanation of the atrophy; but the arguments in support of this view do not appear to me to be sufficient.

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[1] *Charcot*, Leçons sur les Maladies du Système Nerveux. Paris, 1872–3.

[2] *Friedreich*, Ueber progressive Muskelatrophie, etc., 1873, p. 163 et seq.

From the foregoing observations it appears that our information in regard to the significance and mode of origin of the trophic disturbances that occur in neuralgia is very imperfect; but since these questions can now be put in a precise form, their solution, however insoluble they have hitherto proved, may be expected.

Concomitant Psychical Symptoms.—These are rare in neuralgia, the intellectual processes seeming to be scarcely ever affected unless the neuralgia is due to disease of the central organs; it is only during the most severe paroxysms of pain that the mental powers, and in particular the expression of the will, are transiently interfered with. Even in severe and protracted cases no dulness of the intelligence or of the powers of thought is observable, and those in which, by the never-ending pain, serious mental disturbance (general irritability, melancholy, disposition to suicide, and the like) is produced, appear to be comparatively rare. Psychical disturbances, therefore, are quite subordinate features of the disease, and are for the most part only to be regarded as chance concomitant symptoms of neuralgia.[1]

The same may be said in regard to general disorder of the health, for in many instances this is entirely absent, at least so far as it can be considered a consequence of the neuralgia. As a rule, there is no increase of the temperature, no disturbance of the sensorium, no disorder of the appetite or digestive powers, nor is there any loss of weight, and the patient may preserve a blooming aspect for years. In serious cases, however, the long-continued and severe pain does at length impair the general health, the affections complained of being referable to disturbance of the digestive functions, and to the disorder of the organs ministering to blood formation and to the general nutrition, to the incapacity to take in sufficient supplies of food, and to the want of sleep. Thus we observe in those who are the subjects of severe facial neuralgia, at periods when exacerbations of the disease occur, that they not unfrequently lose weight and assume an unhealthy cachectic appearance. Physiological experiments made by Mantegazza,[1] to determine the influence of violent pain on animals, show that it produces loss of appetite, dyspepsia, nausea, arrest of the digestive process, vomiting, and diarrhœa; and, if continued, great weakness and considerable loss of weight, with greater sensitiveness to all injurious agencies. Quite in accordance with this, we find those who are affected with very severe neuralgia suffer from dyspepsia, want of appetite, coated tongue, pallor and anæmia, with cachectic aspect, weak pulse, cold extremities, depressed, excitable states of the system, great sensitiveness to external injurious influences and the like, and there is every reason for believing that these phenomena occur in consequence of the violent action of severe pain upon the nervous system, and through it upon the general health; though, of course, these sequelæ of neuralgia must be clearly distinguished from those general disturbances of nutrition which lie at the root of many neuralgiæ, and owe their origin to totally distinct causes.

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[1] See, however, a remarkable case recorded by *Descot*, in Bretschneider, p. 271, in which serious psychical disturbance occurred in a case of traumatic neuralgia.

Paroxysms and their Causes.—A very marked as well as remarkable feature of neuralgia is its occurrence in separate attacks or paroxysms. This is a pathognomonic characteristic, and is included by almost all authors in their definition of neuralgia. Its periodicity has been especially dwelt on by Anstie.

Nothing remains to be added, in regard to the individuality or distinctness of the paroxysms, their duration and frequency, the regularity or irregularity of their return, the circumstances occasioning them or preventing their appearance, to what has been already stated. It is impossible to give any explanation of the paroxysmal character of the pain in neuralgia so long as our knowledge of the molecular actions taking place in nerves is so obscure and imperfect as at present. The first thing that suggests itself is the *exhaustion* of the nerves that must necessarily occur; and it may be said that if the sensory nerve, owing to strong excitation, be thrown into a paroxysm which ends in exhaustion, the pain ought to cease coincidently with the state of exhaustion. The anæsthesia so frequently observed in the region of distribution of the neuralgically affected nerves agrees very well with this view, as does also the fact that genuine paroxysms occur in neuralgiæ caused by visible anatomical disease, as carcinoma, neuroma, and the like (Trousseau), and in which we should on *à priori* grounds expect the pain to be continuous. Nor is the fact that sensory excitability continues in many instances, after the termination of the paroxysm, altogether opposed to this view, for the ordinary sensory stimuli may act on other parts than do the stimuli causingt he neuralgia, and the capability of transmitting sensations through the neuralgically affected part may be preserved, whilst this part may be itself rendered temporarily unexcitable by exhaustion. This explanation, however, is not quite satisfactory, nor is it in all probability applicable to all cases; it is possible that variations in the intensity of the stimuli, and their periodic occurrence and disappearance, may likewise constitute causes of the periodicity of the pain, but we know scarcely anything definite upon this point.

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[1] *P. Mantegazza*, Dell'azione del dolore sulla digestione e sulla nutrizione, Gaz. med. ital. lombard. 1871, Nos. 6 and 7. Abstract in Virchow and Hirsch's Jahresbericht for 1871.

The electrical relations of the sensory nerves in neuralgia have received but little attention. It is commonly stated, though the fact has no further significance, that the *points douloureux* also are more sensitive to electrical stimulation than other parts, and the same is true of the sensitive vertebræ, and of the sensibility of the cervical sympathetic. In testing the sensitiveness to electrical stimuli in nerves affected with neuralgia, sufficient care has not been taken to distinguish between the electrical sensitiveness of the nerve trunk, and the region of the skin to which it is distributed. The same conditions

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exist in the case of the skin as are met with in testing for other kinds of sensibility ; if hyperæsthesia exists, there is also augmented electrical (faradic and galvanic) sensibility ; if anæsthesia be present, the electrical sensitiveness is diminished.

Benedict was the first to make any precise observations on the greater or less sensitiveness of the nerve trunks to electrical excitation, as well as to draw from thence definite conclusions in regard to the seat and nature of the disease causing the neuralgia. In a more recent paper[1] he has thrown a doubt upon these statements. The difficulties in the way of testing the reaction of the nerve trunks to electricity are very great.

In regard to the electrical relations of mixed nerve trunks in neuralgia, Eulenburg[2] makes some remarkable statements, for he believes he has observed certain anomalies of the laws of contraction in response to galvanic stimuli through motor nerves so that there has even been an absolute inversion of the normal formula ; he has also seen quantitative changes in the galvanic and faradic excitability of the motor fibres. I have not, up to the present time, seen anything of this kind. At the same time the investigation of the reaction of nerves to electrical excitation has not hitherto proved of any assistance in determining th, pathology of neuralgia.

Course and Duration of Neuralgia.

In regard to the course of neuralgia we can here only make a few general observations. Some cases run an *acute course* easting only a few days or weeks, the disease terminating after a short series of more or less violent paroxysms, and this happens for the most part in the slighter rheumatic forms. In other instances the disease is chronic, lasting for weeks or months, and even, if the successive and frequent relapses be included, for years ; in such cases the course is very variable, periods of exacerbation alternating with periods of remission, and even of complete intermission, though recovery ultimately takes place ; this happens, for example, in the anæmic forms of the affection, in those occurring about the period of puberty, and in those associated with hysteria.

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[1] Wien. med. Presse, 1872, No. 21.

[2] *Eulenburg*, loc. cit., p. 154.

Lastly, there are some *incurable cases.* In these the neuralgia is persistent throughout life, though doubtless with intermissions of greater or less duration, and with considerable variations in intensity. These are the terrible cases of *tic douloureux*, which last for twenty or thirty or more years, and those of sciatica and intercostal neuralgia, which never entirely leave the patient. Amongst these may also be enumerated many cases proceeding from organic causes, with well-marked hereditary predisposition, and those accompanying premature old age and vascular degeneration. The chronicity and incurability of neuralgia is often characterized by the term "habit" (Habituellwerden), by which is meant that there is some change in the sensory nerves, which maintains the neuralgia when the cause has disappeared. The expression, however, explains nothing beyond this.

Terminations.—From the observations that have already been made, it appears that neuralgiæ may either be cured or remain incurable. The larger number of cases no doubt terminate in recovery ; it is difficult, however, to give exact data upon the point, because we have no means of ascertaining the further course and the frequency of relapses. The statement made by Valleix, that of 182 cases recovery occurred in 139, appears to represent rather a high proportion, which, however, is explained by the circumstance that Valleix excluded all neuralgiæ caused by evident organic disease. · The mode of termination of the disease depends essentially upon its cause ; if this can be removed recovery takes place; if not, it remains incurable. The result may also be influenced by the general condition of the patient, by his age, general state of health, mode of life, etc. (see Prognosis). Relapses are of remarkably frequent occurrence, and are attributable partly to the general diathesis of the patient, which in most cases remains persistent, and partly to the fact that when the nerves have once been affected with neuralgia, a local tendency to the disease is left which leads to renewed attacks of the disease on exposure to the slightest exciting cause.

Amongst the more accidental terminations of neuralgia are anæsthesia of the skin, paralysis and atrophy of muscles, etc., which are not direct consequences of the neuralgia, but only sequelæ and co-effects of the same causes which have led to the occurrence of the neuralgia; in this way the gradually increasing pressure of tumors, the cicatricial contraction of connective tissue hypertrophied in consequence of neuritis, progressive central disease and degeneration, may, after having first caused neuralgic troubles, lead ultimately to anæsthesia and paralysis.

A fatal termination has scarcely ever been observed in neuralgia. No instance can be adduced in which the violence of the pain caused death, and this can only be conceived to happen from the impairment of digestion and nutrition consequent on the severity of the pain. On the other hand, the diseases lying at the foundation of the neuralgia—such as cancer, bone disease, central affections of the nervous system—may very well occasion death.

Diagnosis.

In making the diagnosis of an attack of neuralgic disease, it is not sufficient to establish the fact that we have to deal with that complex of symptoms which we call neuralgia; for this is comparatively easy to ascertain, and such determination is in many instances quite insufficient to enable us to select an appropriate line of treatment; the diagnosis ought to include the precise determination of the seat of the lesion in the sensory nerves, the causes of this lesion, and the kind and nature of the anatomical changes that may be present.

In the first place, however, it is requisite to determine *whether neuralgia is really present*, whether that characteristic group of symptoms to which the term neuralgia is applied is before us; this determination is entirely independent of the absence or presence of palpable lesions, neuralgiæ occurring both with and without them. As a rule, it is not difficult to recognize the group of symptoms constituting neuralgia. Cases, however, do occur in which a doubt may arise, and painful diseases that are not really neuralgia are constantly mistaken for it. Moreover, neuralgic, like other subjective nervous symptoms, often present the most surprising and exceptional forms, attributable for the most part to idiosyncrasy, and thus place great difficulties in the way of an exact diagnosis. These difficulties may, however, in general, be surmounted by recollecting that the following are essential and characteristic symptoms of neuralgia:

1. That the pain is limited to a definite nerve path, either trunk, branch, or area of distribution, and that it is usually confined to one side.

2. That the pain is, without any obvious reason, either intermitting, or at least distinctly remitting, in character.

3. That the pain presents very peculiar characters, and is extraordinarily acute.

4. That there are certain spots in the course of the nerve, or in the area of its distribution, that are very sensitive to pressure (points douloureux).

5. That the pain is associated with those sensory, motor and vaso-motor, and secretory phenomena which have been already described.

6. That the pain is unaccompanied by any inflammatory or local symptom, or any general disturbance of health at all corresponding with the amount of subjective disorder.

The diagnosis of neuralgia must practically rest on these points; the following may also be ascertained: that the patient suffers from a distinct hereditary or congenital neuropathic diathesis; or that he has previously suffered from some other neuralgia; or that exhausting influences augment the severity of the disease; or that he has been exposed to some well-known and common cause of neuralgia, such as malaria, catching cold, external injury, syphilis, etc. Under these circumstances the diagnosis of neuralgia is greatly strengthened. If all, or the greater number of these symptoms and characters of the disease be present, the diagnosis presents no difficulty; but if several of them are absent, or if they are masked by others, or if similar phenomena caused by other disease are present, an error in diagnosis, as daily experience teaches us, may easily be made. The painful affections which are especially liable to be mistaken for neuralgia are the following:

Muscular pain, muscular rheumatism, that is to say, all those painful affections that are localized in the muscles, and which are known under the names of lumbago, pleurodynia, torticollis, and the like, and which arise either from catching cold or from over-exertion of the muscles. Attention to the following points will readily enable a diagnosis to be established between these "myalgiæ" and the various forms of neuralgia: in the first place, muscular pains correspond to the position of a muscle, and not to the course of a nerve; myalgia, again, is fixed in some definite circumscribed area; it does not usually occur in paroxysms, and is caused and increased by every contraction of the affected muscle; local sensitiveness is only present over the extent of the affected muscle and its tendons.

Spinal irritation, so far as this term is now used to indicate a definite group of symptoms, presents numerous points of similarity to neuralgia, and in many cases it is a matter of taste whether such a group of symptoms shall be characterized as neuralgia or as spinal irritation. The latter is generally an hysterical phenomenon, and is considered to be present when various kinds of wandering neuralgiform pains occur coincidently with great sensitiveness to pressure of one or more of the spinous processes. There is usually more or less concomitant hyperæsthesia of the skin and of the internal organs. The differential diagnostic point lies in the migratory character of the pain in spinal irritation. When the pains are limited to a definite nerve, and there is also sensitiveness of one or two spinous processes, we assume the existence of a neuralgia with a well-marked apophysiary point; when the pains wander, leaping from one nerve territory to another, whilst the hypersensitiveness to pressure affects first one and then another vertebra, we speak of the case as being one of spinal irritation. This distinction may be of considerable importance in the treatment.

The pains that occur in chronic alcoholism, or in those suffering from the toxic influence of *mercury* and the like, cannot easily be mistaken for neuralgia, from which they are distinguished by their persistence, their localization in symmetrical parts of the extremities, especially in the vicinity of joints. The *dolores osteocopi* that occur in syphilis are still more readily distinguished from neuralgic pains by their seat, their symmetrical

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position, and their nocturnal exacerbations. It must not be forgotten, however, that genuine neuralgia may be a consequence of syphilis.

The precise determination of the seat of the pain, and especially the determination of the exact height at which the morbid process affects the peripheral or central fibres, is of great importance in establishing the diagnosis of neuralgia, whether in the peripheric course of the fibres, or in the posterior roots, or in the spinal cord, or, lastly, in the brain; and it may also be borne in mind that the neuralgic trophic disturbance may extend over a large area of the sensory nerves.

The attempts that have been made to determine from the existing symptoms the anatomical seat of the disease occasioning the neuralgia, with some approach to exactness, have not hitherto been crowned with the desired success. No doubt in those cases in which a localized cause of disease is demonstrable, it is not difficult to determine the seat of the neuralgic affection, as in gunshot wounds, the entrance of foreign bodies, neuromata or other tumors, and in injuries of bones. Other cases, however, occur in which such apparent objective causes of disease are not present, and we must then endeavor, by attention to the character and the grouping of the symptoms, the presence or absence of certain concomitant phenomena, and from the occurrence of coincident symptoms of disease of central organs, to obtain precise information in regard to the seat of the disease. Unfortunately, the knowledge we at present possess is insufficient to enable us to draw positive conclusions on this point.

Benedict has made some attempts to determine the seat of the neuralgia from the characters and situation of the pain, and is of opinion that in *true peripheral* neuralgia the pain is of a more continuous character (during the paroxysms), that it follows the course of certain nerves, and is never seated in the bones; on the other hand, in central (excentric) neuralgiæ the pain is of a wandering character, does not follow the course of any definite nerve, is particularly liable to affect the bones, and has a well-marked, sudden, lancinating character. Lastly, a third group may be distinguished in which the pain presents the peripheral (continuous fixed) character, but is localized in the bones, and this indicates that the seat of the affection is in the nerve roots, in the cavity of the skull, or in that of the spinal cord. These diagnostic marks may have some value for the differentiation of peripheral from central neuralgia, though it is probable enough that they do not hold in all cases ; definitive observations are still needed here, and still more to determine the exact characteristics of neuralgiæ affecting nerve roots.

It is always expedient in forming our diagnosis to embrace all the facts at our disposal bearing upon the case, and in this point of view it is of special importance to consider the distribution and extent of the pain, and to determine whether one or several branches of the nerve are affected, though such information must always be taken with some reserve. The fact that a definite branch of a nerve is attacked, as, for example, the inferior maxillary of the fifth, without the lingual, the peroneal without the tibial, and so forth, indicates that the seat of the disease is not to be sought for in the common nerve trunk. The possibility of isolated disease of the affected fibres in the central organs is, however, not to be overlooked. The conclusions that have been drawn from the excentric distribution and the anatomical localization of the pain in regard to the peripheral or central seat of the neuralgia, have consequently only a conditional value, and such conclusions are further affected by the possibility that the distribution of the pain may be due to irradiation.

From this point of view, also, it is important to attend to the numerous concomitant phenomena that occur in neuralgia. In order to recognize the *peripheric* seat of the neuralgia, the concomitant motor and vaso-motor phenomena, which have already been pointed out as having a certain importance in establishing the diagnosis, should be considered ; and these would possess a much higher value if only our knowledge of the anatomical and physiological relations, especially of the vaso-motor nerves, were more complete. The coincident occurrence of motor and vaso-motor disturbances, when these are limited to the region of the neuralgically affected nerve, tells strongly in favor of the neuralgia being of peripheral origin, since it is only in the peripheral nerves that the three sets of fibres run together. At the same time the possibility must always be kept in view that these disturbances may originate by reflex action, and that they may occur coincidently in consequence of influences proceeding from the central organ. Caution should therefore be exercised in founding the diagnosis on this kind of evidence, and the following observations may be made in respect to it:

Motor disturbances indicating irritation, in neuralgia of mixed nerves, are only to be held to point to a peripheral origin of the disease when they are very precisely limited to the motor fibres running in the affected nerve; otherwise the possibility of their being reflex phenomena permits no certain conclusion to be drawn. Paralysis of motor nerves in the area of distribution of the affected nerves tells much more strongly in favor of the peripheral seat of the disease, since this kind of paralysis does not readily originate by reflex action; in such a case, however, it must be tolerably well settled that the disease is not of central origin.

Paralytic phenomena affecting the vaso-motor nerves are very uncertain symptoms on which to found the diagnosis, since they may originate both directly and by reflex action; and they can only be considered to furnish evidence of the peripheric seat of the affection when they occur coincidently with motor paralysis and in a definite area. Vaso-motor phenomena indicating irritation (spasmodic contraction of vessels) may be regarded as decisively in favor of the peripheric seat of the disease, when they are localized in the particular area of the skin corresponding to the affected nerves. Our want of information in regard to the course of the vaso-motor fibres within the peripheral nerve paths greatly increases the difficulty of the subject. From all this it is clear that the phenomena in question do not at present enable us to localize the seat of neuralgia with certainty; earnest endeavors must be made to extend our knowledge in this direction.

In order to recognize the *central* origin of neuralgia the concomitant phenomena indicating the affection of the spinal cord or brain must be carefully considered. These we cannot of course give here *in extenso*. This much is certain, that their recognition demands the greatest care and the widest knowledge on the part of the physician, and that even these are often insufficient, because neuralgic affections are in many cases the very first precursors of the central disease, as, for example, in tabes dorsalis. In such cases we must fall back on the consideration of the nature of the pains and the localization.

Lastly, it is very important to learn the proximate causes of the neuralgia after having determined its seat, that is to say, whether it originates in an impalpable disturbance of nutrition, in neuritis, or in pressure exerted from without, as from periostitis, tumors, and the like, or from some, and which, form of disease of the central organs. The attempt first made by Benedict to distinguish between the several forms of neuralgia (as the so-called idiopathic and neuritic forms, and that arising from compression from without) by means of the symptoms, the character of the pain, the presence or absence of painful points, the sensitiveness to electric stimuli, and the curability, he has in great measure given up. In point of fact it is impossible to make a positive diagnosis from a consideration of the kind and grouping of the symptoms alone. To do this requires a very thorough investigation into all the circumstances of the case, into the causes, the objective evidence and individual symptoms, the concomitant phenomena, the order in which these have made their appearance, their duration, and the general course of the disease. Every experienced physician will admit that in many cases the diagnosis remains obscure for months, and even for years, and indeed may first be disclosed by post-mortem examination. We ought not to omit to state that experience shows that the presence of herpes zoster is strongly in favor of the neuritic origin of the neuralgia. To enter into further details in regard to the symptoms characteristic of the individual forms of neuralgia would here be superfluous.

Prognosis.

In many cases considerable difficulty is experienced in giving a prognosis, since our opinion of the curability and duration of the neuralgia very often rests upon uncertain data.

In general the prognosis of neuralgia is not unfavorable; by far the greater number of cases recover, and even if the very

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favorable statistics of Valleix, to the effect that of 182 cases of neuralgia 139 were cured, is not quite in accordance with ordinary experience, I must still acknowledge that, according to my own observations, about two-thirds of all the cases are sooner or later restored to health (putting aside the possibility of a relapse taking place). There are, however, a certain number of cases which are severe, and run a more chronic course, and there are still others that are really incurable. It remains for us to show how these cases can be recognized, and on what data the prognosis in any given case should be more especially based.

The determination of the *cause* of the disease occupies a prominent position from this point of view, since all those forms of neuralgia which are due to serious organic mischief, as disease of bone, cancer and other tumors, inaccessible cicatrices, etc., lead to a very unfavorable prognosis, and are, in fact, as a rule, to be regarded as incurable. This is also true, though in a less degree, of neuralgia caused by organic lesions of the central nervous system; here, however, the neuralgic affection is sometimes curable, though we are unable to remove the primary disease. The prognosis is unfavorable in well-marked degeneration of tissue, whether senile or otherwise, also if there be a strongly pronounced hereditary predisposition. A more favorable prognosis may be given in cases where the neuralgia arises from anæmia, rheumatic states of the body, and wounds; it is still more favorable in the syphilitic forms, and most of all in those forms of neuralgia that are caused by malaria.

In reference to the *seat* of the disease, as influencing the prognosis, it may be said that peripheral neuralgiæ are in general more promising than those of central origin, which is no doubt connected with the bad forecast that must be made of cases of disease involving nervous centres.

The *particular nerves affected* influence the prognosis to some degree. Neuralgiæ affecting the arm are in general more easily curable than sciatica, and this again is more hopeful than neuralgia of the fifth nerve. Little stress can, however, be laid upon this in any given case, and the prognosis must essentially rest upon other data.

The *age of the patient* is a matter of great importance, and as a general rule it may be admitted that the older the patient the worse the prognosis. In youth, and especially in those neuralgiæ that occur about the time of puberty, the disease is for the most part curable. Those forms, on the other hand, that occur during old age offer a very bad prognosis, and hence it is important in establishing an accurate prognosis that attention should be paid to the indications of senility, commencing atheroma of the arteries, the arcus senilis, etc. The prognosis in neuralgia occurring about the time of the grand climacteric is essentially dependent upon the state of the general health.

The *sex of the patient* is so far of importance, that the prognosis in males is in general more favorable than in females.

With increasing *violence and frequency of the attacks*, as well as with *protracted duration* of the whole disease, the prognosis becomes more unfavorable.

The occurrence of certain *complications*, as of well-marked anæsthesia, paralysis, atrophy, etc., renders the prognosis worse. Lastly, the success or failure of certain modes of treatment, the possibility of adopting energetic measures, the state of the general health, and the like, are points that must be regarded in forming the prognosis, for it is only by the consideration and correct appreciation of all the circumstances of the case that errors can be avoided.

So far as life is concerned the prognosis is almost absolutely favorable, since it is only in exceptional cases that death is attributable to neuralgia. In regard to the removal of the most trying symptom, pain, the prognosis is so far favorable, that with the means we at present possess we can, to some extent at least, in most cases alleviate the sufferings of the patient and render them supportable.

Treatment.

Neuralgia is one of those diseases for which the services of the physician are most earnestly solicited and most gratefully acknowledged. The intensity of the pain leads the patient to resort speedily to medical assistance, and the physician is, for the most part, in a position to afford the desired relief, even if he be not always successful in effecting a complete cure. He should, however, be thoroughly acquainted with, and possess a complete mastery over, all the resources for relieving pain, which have, undoubtedly, been increased in number of late years, and at the same time rendered more precise. We therefore consider it to be our duty to give in this place a full account of the therapeutical measures to be adopted in neuralgia, to which we may be able to refer in speaking of the different forms of neuralgia, and thus avoid repetition.

Considerable difficulties stand in the way of giving, as is now generally preferred, a methodical account, arranged according to the several indications, first, because many remedial measures are applicable to several indications, and must thus be mentioned in different places; and secondly, because we are here dealing with a symptom, and hence the *indicatio morbi* is coincident with the *indicatio symptomatica.* The anatomical changes, the removal of which otherwise forms the object of the *indicatio morbi*, must here be treated under the *indicatio causalis.* If, therefore, we follow the usual arrangement, we do so only on account of its synoptical value, and we are well aware that various objections might be urged against it.

a. *Prophylaxis.*—This is of considerable importance in preventing the occurrence of neuralgia. Prophylactic measures should in the first place be adopted *by those who are predisposed* to the disease, and they are of special value to the members of those families in which the disease is hereditary. The family physician may do much in preventing the development of the affection by timely care and attention.

Good diet is of primary importance; it should be abundant, and should consist of such nourishing substances as meat, bread, eggs, and especially milk and fruits, given in combination with cod-liver oil, and no apprehension need be entertained that such diet will prove too strong or stimulating. In the next place, regular and systematic exercise, in the form of gymnastics, walking, boxing, fencing, swimming, and calisthenics should be regarded as an essential correlate of the abundant supplies of nourishing food, as it powerfully contributes to the strengthening of the nerve system. Exercise, however, should not be excessive, and should alternate with a due proportion of rest. A sufficient amount of sleep, especially during the period of youth and development, is very requisite; Anstie indeed considers that even ten hours is by no means too long. There should be also free exposure to fresh air, whilst bad air and long hours in school should be avoided. No stimulants of any kind should be taken, either in the form of spirituous liquors, or of coffee or tea, and these dietetic measures should be assisted by washing in cold water, and bathing, especially river and sea bathing. The sexual impulse should be carefully restrained, which may in great measure be accomplished by keeping the child pleasantly occupied, by preventing the reading of improper literature, and by a careful selection of companions. Onanism or premature sexual excesses should at all costs be prevented, however difficult this may appear in many cases. Lastly, the whole education must be directed to the mental and physical development of the individual; there should be no superfluous loading of the mind with useless knowledge, but the mind should be directed to earnest, systematic, and yet interesting study. No cultivation of vanity or ambition, no strong religious stimulus should be permitted; there should be no frivolous or useless reading, no attendance on bad superficial theatrical pieces, but true earnest attention to poetry, music, and art. To the thoughtful physician these suggestions will suffice.[1] In many families an excellent basis exists for these measures, but they require to be very thoroughly carried out for satisfactory effects to be produced.

Secondly, prophylactic measures may be adopted by those *who have already suffered from neuralgia*, and are desirous of preventing a relapse. In such cases all those influences which are known to be harmful must be carefully avoided, such as exposure to cold, insufficient or indigestible food, and all mental or bodily over-exertion, especially at those periods which are otherwise favorable to the occurrence of neuralgia, as at puberty, the change of life in women, etc. Those who are anxiously engaged in business should also take some relaxation from time to time, every summer seeking repose and fresh air in some pleasant place. In almost all cases it will be found advantageous to gradually make the patient less sensitive to external injurious influences by a careful and systematically practised process of hardening, it being left to the judgment of the physician in each case what amount of exercise should be taken, and what baths, cold affusions, etc., are likely to prove the most advantageous.

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[1] See, upon these important points, the sound advice given by *Anstie*, loc. cit., p. 212 et seq.

Lastly, prophylactic measures may be adopted with a view of *preventing the paroxysms* in those who are actually suffering from neuralgia. The means best adapted for this purpose are rest, as complete as possible, of the affected part, so that where the patient suffers from facialgia he should neither masticate nor speak, and where from sciatica, he should not walk ; avoidance of exposure to cold and wet, and to draughts of air, as well as (in facial neuralgia) to the bright light and heat of the sun, are important. Care should also be taken to avoid mental excitement, disturbances of the digestive organs, and, speaking generally, all those injurious influences which the patient has learned to recognize as circumstances that are likely to induce the paroxysm.

b. *Indicatio causalis.*—Special attention must always be paid to this, since its fulfilment is in many cases the fundamental condition of recovery. The coarser anatomical lesions fall under this category, since they constitute the immediate cause of the symptom "pain," which in the present state of our knowledge we regard as the chief condition of neuralgia.

The simplest causes of neuralgia are apparently *wounds, and the presence of foreign bodies*, though in many cases no history of these occurrences can be obtained. When the position of the parts allows of it, surgical assistance may be resorted to, and is sometimes crowned with brilliant success, as in the excision or correction of cicatrices, removal of foreign bodies, resection and reposition of crushed or torn nerves, etc. Similar proceedings may be adopted in cases where the neuralgia is due to *mechanical causes*, as the pressure of tumors, compression of nerves from periostitis, hyperostoses, hypertrophied callus, hardened fecal masses, etc. Whenever practicable, these compressing bodies must be removed by surgical or other means, and a cure is thus usually effected. This is especially the case in neuromata and tubercula dolorosa. If surgical interference is impracticable, other measures must be tried, as the use of such resorbents, antiphlogistics, baths, derivatives, etc., as may be appropriate to the particular case, to lessen the volume of the compressing tissue, and to cause its disappearance. Experience, however, unfortunately teaches us that little is to be hoped for from such measures.

If the neuralgia be due to *congestion* or *stasis of blood* in the vicinity of a nerve, the ordinary means used for removing these conditions, as local abstraction of blood, placing the part in an appropriate position, derivation by the bowels, etc., are usually successful. Sciatica, when kept up by stasis in the hemorrhoidal vessels, is often relieved in a striking manner by such means.

Those *organic diseases of the central organs*, which are accompanied by neuralgia, such as cerebral tumors, abscesses in the brain, meningitis, tabes, myelitis, etc., must be treated in the usual manner, for the particulars of which the reader is referred to other parts of this work.

If neuritis be clearly the cause of the disease (*vide* Neuritis), antiphlogistic measures are indicated: abstraction of blood, the application of cold, derivations by the skin and intestines, and at a later period the employment of resolvents and galvanism.

When neuralgia is attributable to *rheumatism*, good results are often obtained by the establishment of active diaphoresis at an early period of the disease, and this is best accomplished by vapor baths, with local antiphlogistic measures and derivation by the skin, as by sinapisms and blisters. The older physicians (Eisenmann) expressed themselves strongly in favor of colchicum and opium in these cases. In chronic rheumatic cases, the indifferent (non-saline) thermal baths of Wildbad, Teplitz, Gastein, Baden-Baden and Wiesbaden, are very useful, and the cold-water cure and electricity may also be employed. Some benefit may also be obtained from the administration of iodide of potassium, and from wintering in a mild climate.

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The energetic treatment of the *neuropathic diathesis*, which so often lies at the foundation of neuralgia, is also of much importance. This must be proceeded with on general therapeutic indications, a detailed account of which would lead us too far. It may, however, be here briefly remarked, that the so-called nerve-invigorating method of treatment must be adopted in the widest signification of the term, particular attention being paid to the diet and general habits of life, to sufficiency of sleep, to regular exercise in the open air, to the use of cold water in washing or in baths, and to sea-bathing, whilst residence in a mountainous district may also be recommended. In addition to all this, the remedies appropriate to the particular case—as quinine, iron, nervine tonics, phosphorus, strychnia and others, and ultimately, resort to medicated springs—must be employed. In these cases careful attention to the idiosyncrasies of the patient, appropriate selection of the method of cure to be adopted, and long persistence in it, are alone likely to prove effective, as it is notorious that a very large number of cases of neuralgia are attributable to the existence of a nervous diathesis. The physician who undertakes the treatment of these cases should make himself familiar with the most minute details of all the means that are capable of strengthening the nervous system. If *hysteria* be unmistakably present, anti-hysteric remedies may be prescribed in addition to other things.

Anæmia, in its manifold forms, is likewise of importance, as constituting a cause of neuralgia, and the high esteem in which the ferruginous preparations are held is essentially due to the circumstance that so many neuralgiæ are due to anæmia. Here, also, it is impossible to enter into details; it is enough to say that the most diverse modifications in the generally received method of treatment of anæmia and chlorosis may here be requisite, but there can be no doubt of the value of iron, accompanied by abundant food; when the former proves of no avail arsenic is often serviceable. Anstie recommends a combination of solution of chloride of iron[1]—from ten to fifteen drops—with a fortieth of a grain of strychnia, taken several times a day, as being particularly effective in the anæmic neuralgia of young women.

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[1] The U. S. Solution nearly corresponds to the "Stronger Solution" of the British Ph.

In *dyscrasic* and *cachectic conditions*, as for example, in scrofulosis, tuberculosis, gout, etc., the usual dietetic and pharmaceutical measures that have been found effective in these diseases must be adopted. If *syphilis* be regarded as the cause of neuralgia, the mercurial preparations and iodide of potassium, according to the stage of the disease, are indicated, and usually prove very effective. In those who are suffering from the *toxic influence of alcohol*, *mercury*, *lead*, or other substances, the appropriate remedies must, of course, be energetically employed.

Results of a highly satisfactory nature are brought about by treatment in cases of *malarial neuralgia*. In those cases of periodic neuralgia that are distinctly traceable to malarial infection, a few full doses of quinine have an almost magical effect; and where this fails, as is occasionally the case, recourse must be had to arsenic.

Indicatio morbi et symptomatica.—We associate these together, since most of the remedial measures that will be mentioned act in both directions, whilst we are at present wholly unable to make any definite statements in regard to the essential nature of the malady, and must still consider it as a symptomatic form of disease, in which the treatment of the principal symptoms coincides with that directed to the disease itself.

We here meet with an enormous number of remedies that have been popular, and some of which still continue to enjoy the reputation of being anti-neuralgic. A critical sifting of the abundant material that has been handed down to us from remote periods was necessary, and this has been in great part accomplished during the last ten years. Modern investigations have reduced the anti-neuralgic remedies to a few groups; the rest have fallen into disuse to be only now and then tried in desperate cases.

Here, perhaps, is the best place to append a few observations upon the *diet* and *mode of life* that should be adopted by neuralgic patients, since the careful regulation of both must in all cases form an essential part of the treatment, and since in and by themselves they not unfrequently fulfil several of the more important indications. The remarks that we shall here make are, of course, chiefly applicable to the more severe cases.

In the first place, the patient should be most careful in maintaining both mental and bodily rest, no violent exertions should be made, and all mental excitement must be avoided; if possible, business for a time should be entirely given up. In the next place, all means should be adopted to give the patient complete rest, and especially a large amount of sleep; if necessary, this must be obtained by the use of medicines; amongst them chloral, especially in combination with a little morphia, is the best. Great regularity in the mode of living must be particularly insisted on; attention being paid to the due alternation of exercise and rest, to regularity in the hours at which the meals are taken, and to a certain amount of rest after meals. Moderate exercise in the open air in fine weather, or mere exposure to the air in those who are very delicate, is very useful. The food (with due regard always to individual peculiarities) should be good and abundant, especially in very young or aged persons. Anstie can scarcely sufficiently extol the advantage to be derived from the consumption of even a larger supply of food than is necessary for a healthy person; and this has been found particularly beneficial in all chronic neuroses. A large supply of fat with the food is of special value when taken with abundance of meat, milk, eggs, and bread. On this account the continued use of cod-liver oil in large doses is strongly recommended; where this cannot be endured, attempts may be made to replace it by the free use of cream, butter, olive oil, cocoanut oil, and the like. Unfortunately, neuralgic patients have often a bad appetite and a strong aversion to certain kinds of food, while their stomachs are not in a condition to bear the more liberal diet recommended to them; patience and humoring on the part of the physician, and with good-will on the part of the patient, the augmented quantity of food may in many cases be taken. The use of spirituous liquors can only be allowed when they are taken as food at meal-times; in this way there is no objection to a little good wine or strong beer; on the other hand, the patient must avoid the excessive use of alcohol; it should by no means be taken in intoxicating doses.

The advantages of uniformity of temperature should always be impressed on neuralgic patients, and the clothing should be carefully adapted to give protection against sudden cooling and catching cold, but it should not be too warm; on the contrary, some efforts should be made to harden the skin by sponging with cold water, friction, and the use of baths, at first of a warm temperature, and subsequently becoming colder. As a general rule, the use of baths, as regiminal means of treatment, should not be neglected. Anstie recommends also the Turkish (hot-air) baths, with subsequent cold douche on the back, as a very effective means of hardening the constitution. Finally, the patient must practise the prophylactic measures enumerated above against the occurrence of the paroxysms. Modifications and extensions of this plan of treatment, which cannot here be further discussed, must, of course, be made in accordance with the idiosyncrasy, the social position and mode of life of the patient, and our knowledge of the etiology of the disease.

The *specific remedial measures*, adopted for the cure of neuralgia, all have for their object to neutralize the supposed trophic disturbances of the nerves, and thus to remove the principal symptom of neuralgia, the pain. How this is effected by the means employed we are unable at present to offer any satisfactory opinion, because we are ignorant of the essence of the pathological trophic disturbances, the finer details of the mode in which neuralgic pain originates. The theories ordinarily advanced regarding these modes of operation (as by removal of anatomical changes and disturbances of the circulation, by warding off every abnormally strong excitation, by diminution of the pathological excitability of the nerves, etc.) are, no doubt, reasonable, but still have too small a foundation on solid facts to render it of any service to the practitioner to consider them more closely. We, therefore, at once proceed to the consideration of the several remedies themselves.

There are three groups of remedies that occupy the foremost place in the treatment of neuralgia, as being the most effective. 1. *Electricity* in its various forms. 2. The *narcotics*, especially when applied hypodermically; and, 3. Certain *nervine specifics*, which experience has shown to be useful in many cases. The

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remedies at our disposal are not, however, exhausted with this list, for in the application of cold, the use of baths, the employment of derivatives, and in surgical interference, we possess other means which, however, are of secondary value as compared with the three groups just mentioned.

1. *Electricity* has recently become the most important remedy in the treatment of neuralgia, from the brilliant success that has attended its application in all the different forms of the disease. Its mode of action is still obscure, though it probably varies in different instances. It diminishes the excitability of the sensory nerves, it takes away abnormal stimuli from them, it modifies their nutrition, allays hyperæmia and inflammation, and lastly acts as a derivative, and thus fulfils several indications. The methods of applying it are manifold, especially since the reintroduction into practice of the galvanic current by Remak. The results of electro-therapeutical treatment are in no other disease so certainly established as in neuralgia. Of the two kinds of electricity now in constant use, the galvanic current is found to be more active and applicable to a greater variety of forms of the disease than faradic electricity.

Faradic electricity is chiefly useful in peripheric neuralgiæ, when the nerves can be reached by the current, and in cases where no remarkable anatomical change, as neuritis or the like, is present, and thus especially in the so-called purely idiopathic or "habitual" neuralgiæ. It has also somewhat of a derivative action, as a stimulant to the skin.

The *galvanic current* has at least the same action upon peripheric neuralgiæ, whilst, in addition, it is very effective in the central and deep-seated forms of the disease (spinal and cerebral neuralgiæ, and neuralgiæ of the roots of nerves). Moreover, by its "catalytic" effects, that is to say, by its influence on the vessels, upon exudations and the processes of nutrition, it exerts a wide influence on those neuralgiæ which are uninfluenced by the faradic current.

The special indications for the employment of this remedy cannot, however, as yet be given with precision, since most of the facts have been discovered empirically, and still require much corroboration. As a matter of course, electricity will not cure all forms of neuralgia; it cannot even be accounted a sure palliative in all neuralgiæ, but is surpassed in this by the narcotics. This is particularly true of neuralgiæ which are due to gross anatomical changes, and of those caused by anæmia and the different forms of poisoning, so long as these causes continue to act.

On the other hand, electricity, and especially the galvanic current, has been found to yield the most gratifying results in the so-called idiopathic neuralgiæ, in most of those which are due to a rheumatic or neuritic process, and which have become habitual, and finally in a certain proportion of the excentric neuralgiæ. Indeed the results are sometimes astonishing, so quickly do they appear. In other cases, however, it is necessary to persevere in this mode of treatment for a long time, and with great regularity, before a cure is effected.

The results that various electro-therapeutists agree in stating they have obtained are extremely favorable. In the well-known text-books of electro-therapeutics, as in those of Althaus, M. Meyer, Rosenthal, Benedict, Beard and Rockwell, and Duchenne, we have the most indisputable evidence on this point. I can myself corroborate these favorable statements in the most complete manner, and in earlier papers[1] have given an account of the favorable results I have obtained in a great number of different cases.

The modes of applying electricity can here be only shortly given, and the reader is referred to the ordinary handbooks of electro-therapeutics for further details.

There are two methods of applying *faradic electricity:*

a. By conducting a strong current of the secondary spiral, for a few minutes, through the nerve, by means of moist electrodes, one of them being placed on the nerve trunk as near as possible to its central origin. This plan must, for the most part, be frequently repeated.

b. By producing energetic irritation of the skin with an electric brush, or by means of an electric moxa (Duchenne, M. Meyer), in the region of distribution of the nerve, at its point of emergence, and over the *points douloureux.* This method, which is often very effective, ought properly to be included under the head of derivative means (see below).

The application of *galvanic electricity* is especially intended to modify the nutritive processes taking place in the nerve, to produce the so-called catalytic effects, and to lower the irritability of the nerves. These objects are best fulfilled by the use of the continuous currents. The question which is now so warmly discussed by electrotherapeutists as to how these are to be applied, whether according to the polar or the direction method (Richtungsmethode), requires first to be definitively settled. The results, however, of both plans seem to be equally good. In the polar method the anode is best applied continuously with a current that gradually increases and diminishes in intensity; the anode is applied first upon the nerve trunk (when possible in the immediate vicinity of the proper focus of the disease) and then upon the *points douloureux*, and the cathode upon some indifferent point. In many instances, the continuous action of the cathode acts better, which is readily explicable on the grounds of its catalytic action. In the direction method, the descending direction of the current is used by preference, and the anode is then to be placed upon the plexus or upon the roots of the nerve, and the cathode upon the nerve trunk and the painful points. The fundamental idea here is therefore always "local treatment." This has especially important results in the treatment of excentric neuralgiæ. This is only effective when the current is applied directly to the brain or spinal cord, but in many cases a treatment directed to the periphery is also requisite, with the object of removing abnormal conditions of excitability in the peripheral nerve paths. The indirect method of treating many neuralgiæ, introduced by Remak, viz., by acting on the sympathetic ganglia, and the "general electrization" proposed by Beard and Rockwell for the treatment of constitutional neuralgiæ, are methods that have not as yet been sufficiently controlled and tested to merit a recommendation.

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[1] Deutsches Archiv f. klin. med., Band III.

In regard to the duration and the frequency of the sittings no general statement can be made. As a rule, they should be short, extending over from two to eight minutes, and repeated daily, or every other day. In only rare instances will it be found necessary to apply the current more than once a day. The strength of the current must, in general, be moderate.

The effects are usually experienced at once, and continue for a variable period, from two or three to twenty-four hours, ultimately after a variable number of sittings becoming permanent. If after a moderate number of sittings, as from six to ten, no appreciable benefit is experienced, the case must, in general, be regarded as one not adapted for the electrical plan of treatment.

2. *Narcotics* (and *anæsthetics*) in the present day constitute very important remedies in the treatment of neuralgia, and cannot be neglected, since they constitute palliative means that are uncommonly certain and agreeable in their mode of action. From this point of view they are unsurpassed by any remedies, especially since we have learnt to introduce them by subcutaneous injections into the system, in very small and at the same time very effective doses. Since this discovery their beneficent action has been extended to a very wide class of cases. Although

numerous experiments have been made with these drugs, little is known in regard to their mode of operation. They were originally prescribed merely as pain-quellers, that is to say, purely symptomatically, and we are rarely disappointed in regard to this part of their action, but experience has shown that many neuralgiæ completely recover under their use, and it is therefore evident that they must act as genuine curative agents. The pain-quelling action is usually referred to the power of changing the condition of excitability, which narcotics exert over both the peripheral and central sensory apparatus. The action on the central organs is the more marked and important, and is that which best explains the powerful palliative influence exerted by the narcotics. As to the mode in which they act as curative agents, the hypotheses that have been advanced are sufficiently unsatisfactory. It is said, for example, that they produce a permanent diminution of the excitability, or in other words some intimate trophic changes in the sensory apparatus ; it is also conceivable that, owing to the sleep obtained and the rest given to the nervous system by narcotics, the trophic disturbances are calmed down, and the path to recovery opened. However that may be, it is certain that in the hypodermic injection of certain narcotics we possess an excellent and certain palliative in neuralgic affections ; and besides, in many forms of neuralgia, and especially in the slighter and more recent cases affecting the periphery, a permanent cure is often effected by these remedies.

By far the most important of the means in question are the alkaloids of opium, and especially morphia, which may indeed be regarded as the chief of those which can be hypodermically injected, since it acts most advantageously and most certainly when thus introduced into the system.

Hypodermic or subcutaneous injection has become so thoroughly popularized that it is unnecessary to enter into any details regarding it. It consists in the introduction of a solution of the remedy into the subcutaneous connective tissue by means of a small Pravaz or Luer's syringe, which is armed with a fine perforated stylet. The most important thing in this method is to actually introduce into the subcutaneous connective tissue a very precisely measured quantity of the fluid; and this, with a little practice, can easily be accomplished without materially inconveniencing the patient. Care should be taken not to inject the fluid into the tissue of the cutis, as this is likely to produce considerable pain and swelling, and even inflammation of the part. The locality chosen for injection is not altogether a matter of indifference. When the general action upon the central nervous system is desired, those parts of the skin may be selected which are thin, and where the subcutaneous connective tissue is loose, as, for example, the temple, clavicular region, anterior abdominal wall, and internal surface of the fore-arm and thigh; but when a decided local action is intended, as is usually the case in neuralgia, the injection should be made as near as possible to the nerve trunk affected, or into the painful point. The advantages of subcutaneous injection depend upon the great rapidity and certainty with which the medicaments are absorbed, upon both the general and local action being obtained, upon the possibility of determining the dose with great precision, whilst at the same time much smaller doses are required, and lastly, upon the digestive organs and appetite being but little or not at all disturbed.

The dosage is best effected by making the solution of such strength that each division of the syringe, or each five or ten divisions, contains a definite fraction of a grain of the remedy, so that the quantity injected may be easily read off. The syringe must, of course, be carefully graduated. The best solvent is distilled water, but more consistence can be given to the fluid by the addition of an equal quantity of pure glycerine. The substance to be injected ought to be first dissolved in the warm glycerine. In regard to the details of the mode of injecting, the precautions that should be taken, the precision of the dosage, the media to be employed, and the general facts in respect to hypodermic injection, the work of Eulenburg [1] may be referred to.

The dose of morphia that may be injected varies from one-thirteenth of a grain to one and a half grains, and it is usual to begin with the smaller and gradually to rise to the larger doses; when the effective dose has been reached it may in general be persisted in for a considerable period, though by degrees the system becomes accustomed to it. Unpleasant, though not dangerous, symptoms are not unfrequently observed, especially when the injection has been made about the head or neck, such as feeling of anxiety, faintness, mental confusion, drowsiness, stupor, nausea, and vomiting. These effects are for the most part only transitory, but it is prudent to prepare the patient for their possible occurrence. Morphia injections are prescribed in those cases in particular where a rapid and certain means of removing the pain is urgently required, but also where it is desired to relieve the sufferings of the patient by palliative means, until a cure is effected by other remedies; and lastly, it is employed as a curative agent, if indeed it may be assumed that morphia can of itself effect a cure. The injection should not be made too often; as a rule, once, or at the most twice a day is sufficient, though in severe cases it may be done more frequently. When it is employed as a curative means, it is advisable for several days consecutively to suppress the paroxysms of pain as completely as possible by means of injections; this appears to promote a cure very greatly. The effects of the injection of morphia are usually very striking; the patient is often relieved from most violent pain in the course of a few minutes, and feels himself a new man, capable of speaking, eating, and moving about. This freedom from pain lasts for some hours or days, according to the violence of the pain and the amount of morphia that has been injected. When large doses of morphia have been taken for a considerable period, the patient becomes so habituated to them that they cannot be pretermitted without producing the most tormenting symptoms; in such cases the quantity must be gradually diminished; if done too rapidly, the patient often falls into a condition of extreme weakness and great general discomfort, which can only be overcome by the free administration of stimulants. Injections of morphia are, of course, contra-indicated in states of great debility, in advanced age, hyperæmia of the brain, organic disease of the heart, etc.

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[1] *Eulenburg*, Die hypodermatische Injection, 2. Aufl. Berlin, 1867.

With the exception of morphia, the other preparations of opium, such as extract of opium, narceine, codeine, narcotine, and the like, are but little employed. They may be safely neglected.

Atropine alone of the other narcotics deserves special mention. It exerts an anti-neuralgic action similar to that of morphia, and may even prove serviceable when this fails. According to Anstie, it is particularly useful in glaucomatous neuralgia, and in neuralgia of the pelvis. It should, however, only be resorted to in cases of necessity, on account of its highly poisonous action, which is perceptible sometimes even when extremely small doses have been administered. The dose for injection ranges from the one one-hundred-and-thirtieth to the one-twenty-second of a grain. Althaus states that the combination of morphia one-sixth, and atropine one-sixty-fifth, of a grain, proves very useful in many cases.

In cases where hypodermic injections cannot be employed (as often occurs in private practice), narcotics must be administered internally; but larger doses are required, and these are both less active and not unfrequently disturb the digestion. Morphia may thus be given in frequently repeated doses of from one-sixth of a grain to one and a half grains, and it may be advantageously combined with moderate doses of quinine. This combination is better borne, and is more effective. The other preparations of opium, as well as atropine and the extracts of belladonna and hyoscyamus, may also be given internally.

But little confidence can be placed in the effects of the *external application* of narcotic remedies. The inoculation and endermic application of the narcotic alkaloids, which were formerly occasionally practised, are now fallen into complete disuse. We cannot, however, afford to dispense entirely with the use of narcotic ointments and embrocations in ordinary practice, since it cannot be denied that their application is often followed by favorable results. Still, too much, especially in serious cases, must not be expected from them. The salve or lotion, when these are employed, must be rubbed into the painful part of the skin or over the affected nerve several times a day, or compresses smeared or moistened with them may be applied, or, lastly, narcotic plasters may be kept applied to the part for some time. Of these it will be enough to mention here salves containing morphia, or extract of opium or of belladonna, and salves with veratrine (one part to twenty), or aconitia (one part to forty), which may be rubbed in till pricking sensations in the former cases, and numbness of skin in the latter, are induced. I have often found a salve effective which contained one part of veratria, and one of watery extract of opium, combined with twenty parts of fat. Trousseau recommends as a good palliative a watery solution of atropine, one grain to the ounce, to be applied by means of compresses, or one of double the strength to be rubbed into the parts several times a day. Extract of belladonna, one part, and four parts of glycerine, and four parts of starch may also be rubbed in and applied on a compress. These and similar preparations will not infrequently give considerable relief to the patient.

Associated with the narcotics, and having a similar action to theirs, are the *anæsthetics*, chloroform, Dutch liquid, nitrite of amyl, etc. Their action, however, is less permanent and less energetic, though in many cases it is sufficiently marked. After their absorption into the blood, they act by producing anæsthesia of the sensory centres ; when applied locally, by producing anæsthesia of the terminations of the nerve, and in part also as rubefacients (derivatives). They may be used, *a*, in the form of inhalations, but in this case they should only be given to an extent sufficient to remove the pain, and not to complete unconsciousness. This may be effected by prolonged but moderate inhalation. They are particularly serviceable in alleviating very violent paroxysms of pain. They may be used, *b*, internally. Thus chloroform or ether, in doses of from ten to twenty-five drops, may be prescribed in some mucilaginous solution, and then serves as a moderately effective palliative. They may also be used, *c*, in the form of an enema, in which both ether and chloroform can be given in doses of from fifteen to twenty minims suspended in starch paste. Thus given, they act in a similar manner, and may be ordered in neuralgia of the sacral plexus, or of the pelvic nerves. Lastly, *d*, they may be prescribed in the form of ointment, or liniment, or in such a way that the ether or chloroform evaporates from the surface, which is best accomplished by means of the spray apparatus, or a piece of wool saturated with the fluid is placed on the painful parts of the skin, evaporation being prevented by means of a watch-glass, or other cover. All these therapeutical measures are applicable when a change in the mode of treatment appears desirable, as, for instance, where it is thought best not to continue the use of narcotics for too long a time.

This appears to be the place to make a few observations in regard to the use of the *hydrate of chloral*. However ineffective this remedy is in cases of severe neuralgia, its well-marked hypnotic action is very useful in many cases, and for this purpose it may be advantageously combined with small doses of morphia, in the proportion, for example, of fifteen grains of chloral to one-sixth of a grain of morphia. Very recently, cases have been reported showing the favorable action of chloral alone upon recent and slight forms of neuralgia.

3. The group of *specific remedies* includes a great variety of remedial measures, the effects of which on the states of nutrition and excitability afford reason for believing that we may, with some confidence, expect them to cure neuralgia (alterantia nervina). These means have in part been discovered empirically, and their value demonstrated by experience; they have also in part been introduced into the treatment of neuralgia in consequence of pharmacological or physiological researches, or for reasons deduced from pathological considerations. In many instances they have been adopted on very insufficient evidence of their efficacy. Experience has not as yet pronounced a final judgment upon all of them, but it is certain that many of them may be regarded as inoperative, and at least superfluous, so that their almost infinite number may be advantageously reduced to a few that still admit of being employed.

The influence of these last is undoubted, and has been satisfactorily established by the testimony of excellent observers; but their mode of action is for the most part entirely unknown. Some certainly act by effecting a general improvement of the nutrition, and of the blood-making organs and processes; others by their influence on the circulation and upon the vessels; others by their modifying directly the molecular relations of the nerves; and others again by effecting changes in the nutrition of the several tissues. Our ignorance of the precise mode of action in any case has led to a purely empirical application of these means, and we are often tempted to resort to them when the preceding groups of remedies have failed or happen to be inapplicable.

In the very first rank amongst specific remedies we must place *arsenic*, which acts not only as an anti-periodic remedy in neuralgiæ of malarial origin, but also as a proper nervine tonic. As is well known, a very remarkable influence upon the general nutrition of the body and the tone of the nervous system is ascribed to it. As Isnard observes, "arsenic is a neurosthenic tonic, it increases the nerve force and restores order to its action when disturbed." It certainly displays a singular curative power over many forms of neuralgia, not only in recent cases, and in those exhibiting regularly recurring or periodic attacks, even when they are not dependent upon neuralgia, but also in chronic cases of purely idiopathic neuralgia which have proved rebellious to treatment. It is especially effective in cases where there is a general nervous diathesis, and imperfect formation of blood. In such cases Fowler's solution may be given in doses of from three to ten drops three times a day, in gradually increasing doses, or the arsenious acid may be given, dissolved in water, in doses of from one-eighth to one-half of a grain per diem, in divided doses. In all instances the use of the remedy should be continued for a considerable period of time. Recently arsenic has been injected hypodermically (Eulenburg), and it must be admitted that this method of using the remedy possesses certain advantages, as I can myself testify from personal experience.

Zinc has been highly praised, but whether given in the form of oxide, or of valerianate, or of sulphate, it must be prescribed in large doses. The indications for its exhibition are uncertain. Oxide of zinc, with extract of hyoscyamus, constitutes the essential constituent of Meglin's pills, which were formerly in high repute.

Phosphorus, in doses of from one sixty-fifth to one-eleventh of a grain has been warmly recommended, especially in anæmic and neurasthenic neuralgiæ.

Bismuth, *nitrate of silver*, *chloride of barium*, *chloride of gold and sodium*, *lead*, *and mercury* are recommended by many observers, but are seldom effectual in curing the disease. Corrosive sublimate is considered to be a valuable remedy in chronic rheumatic neuralgiæ.

The preparations of *iron* are of undeniable value in the anæmic forms of neuralgia, and, by their blood-making properties, fulfil an important indication of treatment. Whether they act directly as nervines is very doubtful, and the carbonate of iron in particular, which was formerly given in such enormous doses, appears to possess no specific action on neuralgia. Anstie,

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however, ascribes a remarkable and specific action upon the nerve centres to the chloride of iron.

Quinine also deserves to be mentioned here, since it has a very decided action on neuralgiæ, even where they are not dependent on malaria. I have found it to be of great service, when given in combination with small doses of morphia (quinine three grains, morphia one-sixth of a grain, several times daily), in recent rheumatic cases. It is highly praised also by Trousseau. Naunyn has recently recommended it in enormous doses (from two scruples to two drachms per diem) in old neuralgiæ, especially if these are referable to malaria.

Strychnia is praised by Anstie, whether given internally or injected hypodermically, and it may be given combined with the solution of chloride of iron.

Bromide of Potassium is extremely valuable, especially in cases where it produces an hypnotic effect. Anstie recommends it in neuralgia attacking those who, whilst otherwise in good health, exhibit a certain restlessness and irritability of disposition, which is often the consequence of insufficient gratification of the sexual passion, as in women condemned to celibacy. As in epilepsy, large doses are requisite.

We must also observe in reference to the *iodide of potassium*, which is useful in so many diseases, that it proves serviceable also in many cases of neuralgia, as in those of chronic rheumatic character, and in very obstinate idiopathic cases.

Amongst animal and vegetable remedial means *turpentine* is the only one that enjoys a certain reputation; it constantly reappears as a successful remedy, its employment being justified by what seem to be very favorable results. The indications for its use are very uncertain, but it may be given with advantage in chronic, apparently hopeless cases, especially of sciatica. It often proves of unquestionable value. About fifteen or twenty minims may be given several times a day, and the best mode of administration is in the gelatine capsules, and the best time for taking them is at meals.

Valerian, *assafœtida*, and *castoreum* may be tried in cases of hysterical neuralgia.

In addition to the several more important groups of anti-neuralgic remedies just mentioned, there are still others that may be occasionally prescribed with advantage. Amongst them is *cold*, which, as is well known, is a very active agent in lowering the excitability of the nerves, and in diminishing the sensibility of the region to which it is applied. This has led to its employment in neuralgia. It is, of course, only effective in those cases where its influence can reach the nerve affected, and, therefore, only when the nerves are superficial. And since cold also exerts a powerful antiphlogistic and anticongestive influence, it may be advantageously applied in neuralgia occasioned by neuritis, hyperæmia, and rheumatic inflammation. Even in neuralgiæ of centric origin, the application of cold to the head or back may perhaps prove serviceable. The modes of application are by means of ice bladders, ether spray, irrigation, cold douches (cold-water cure).

Derivation is an old and well-established remedy in neuralgia. Derivatives were formerly very commonly employed, and are still much relied upon by the public; they may be prescribed in many cases, since the results of their action are often exceedingly satisfactory. By the term derivation is understood the removal of a pathological process or condition of excitation in any part of the body, by the artificial establishment of a similar process in some more or less distant part. The physiological processes set up by this procedure have been but little investigated, and most of the facts regarding them have been ascertained in a purely empirical manner. As derivatives we employ, *a*, strong cutaneous irritation, which effects a change in the sensory excitation and sensation in other nerves, whilst a powerful influence is exerted upon the vaso-motor nerves, causing contraction or dilatation of the vessels; *b*, measures for producing well-marked hyperæmia, by which means the removal of congestion at the seat of disease is effected, with diversion of the blood current to unimportant parts; *c*, measures for producing inflammation, and by this means a "derivation" of the original inflammation and depletion of the blood-vessels; *d*, measures for exciting an abundant secretion, thus leading to depletion of the circulatory apparatus, the reduction of hyperæmia, removal of exudation, etc. Most of these derivatives act upon the skin, but

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a few also act upon the intestines and kidneys. The former are always used by preference in neuralgia.

The electric brush and moxa undoubtedly constitute the most conveniently applied, and at the same time the most energetic cutaneous derivatives. They produce most severe sensory irritation, whilst their action can be graduated at will, and leaves no traces behind it. The results of these applications in neuralgia are most brilliant; the pain is either temporarily or permanently removed; they are especially to be employed in peripheral neuralgiæ, in which there are no appreciable anatomical changes. The actual cautery, moxa, etc., act in many cases in a very similar manner, namely, as powerful irritants of the cutaneous sensory nerves; this is particularly the case with the actual cautery which, when applied to remote parts of the skin, as for example, to the auricle of the external ear, can only be supposed to act in this way. It is notorious, however, that such application not unfrequently relieves, for a time, and sometimes even permanently, severe chronic neuralgiæ, sciatica, for example. In other cases the actual cautery produces its beneficial effects by the subsequent inflammation and suppuration which it causes.

Amongst the rubefacients in most common use are sinapisms, embrocations with turpentine, with chloroform, with ointments containing veratria, with camphorated spirits, and with iodine, and the direct application of moistened nitrate of silver, which have all been recommended, and may be used in the slighter rheumatic and idiopathic cases.

Amongst the vesicants the ordinary *cerate of cantharides* is by far the most commonly employed. It has been particularly recommended by Valleix, as one of the most important means of cure in all forms of neuralgia, and has proved equally useful in the hands of other observers. Its precise mode of action is still obscure, though its effects are doubtless principally due, first, to its stimulating influence on the skin, and secondly, to its vesicating power; Anstie indeed maintains that it acts as a stimulant to the nervous system. The beneficial effects are most marked in recent rheumatic cases, though they are often not altogether wanting even in chronic cases. The best place to apply the so-called flying blisters is along the course of the diseased nerve. Anstie considers their application over the intervertebral foramina, through which the nerves affected emerge, to be particularly efficacious. It is only in very chronic cases that blisters need be kept open for a long time, and they prove of little comparative value in old people.

The other agents which may properly be mentioned in this connection are mezereon, croton oil (used as an embrocation), and suppurating ointments of various kinds, but they are far less effective and much more inconvenient than the blisters. The application and maintenance of issues may be regarded as an antiquated method of treatment.

The filiform douche, acupuncture, the subcutaneous injection of common salt and of nitrate of silver, act as powerful local irritants, and may, as such, be applied to the cure of neuralgia.

Lastly, the *actual cautery* must be regarded as an extremely important means in many cases, although the indications for its use unfortunately cannot be laid down with much precision. It may be applied in chronic and obstinate cases, where no severe organic disease is discoverable. On the other hand, it is much surpassed as a palliative by other remedies. The best means of applying it is by drawing lines with it on the skin.

Derivation by the bowels and kidneys is seldom employed in neuralgia, and then only when there are strongly marked indications for its use. As a general rule, it may be said that derivative means are best adapted for slight and recent cases, especially those of rheumatic and inflammatory origin, and for certain idiopathic forms, and that they act most beneficially on strong and young patients.

Baths and *water cures* constitute additional and important remedial measures in the treatment of neuralgia, and all possible forms have been tried in severe and obstinate cases. The water cures are not only employed to improve the general health (as hot baths, ferruginous, saline, and sea-water baths), but they are also used for the specific cure of neuralgia. The *indifferent thermal baths* are of the greatest service, on account of their calmative action, especially if the temperature be moderate.

In selecting a few of the more prominent ones, we may mention those of Schlangenbad, Pfäffers, Ragatz, Wildbad, Gastein, Wiesbaden, Teplitz, and Leuk. Lastly, the vapor baths, and the so-called Roman-Irish (hot air) baths may be mentioned. It is very difficult in the present state of our knowledge to lay down specific indications for their use, but the cooler thermal baths may be used in the neuralgiæ that occur in persons with over-excited nervous systems, in hysterical cases, and in those of spinal irritation, etc. Baths of a higher temperature are serviceable in cases of neuralgia associated with chronic rheumatism, and in those of traumatic origin (gunshot wounds, cicatricial contractions, and the like), in which there are corresponding motor and trophic disturbances. The thermal baths of high mountain regions may be recommended for old people with commencing atheroma, who require general freshening up and strengthening of the whole system.

Besides the thermal baths, little remains to be mentioned. Mud baths are oftentimes useful. Ferruginous waters should only be taken when there are definite indications for their employment. Cold water cures and sea-water bathing prove serviceable in many chronic cases, but should also be taken in accordance with special indications. Air cures, residence in mountainous regions, and the like, as well as whey cures, grape cures, etc., must be prescribed in accordance with the causal indications.

An ultimate and in many cases obviously very rational means of treatment of neuralgia consists in *preventing the conduction of the painful irritation to the brain.* This plan can, of course, only be practised where it is possible to interfere with the conduction between the seat of the disease and the brain centres. The interruption of the conduction in the centripetal fibres may be effected—

a. *By compression of the nerves*, in which case the compression must be sufficiently energetic and sustained to arrest sensory conduction. This is a very questionable and dangerous method, owing to the fact that it is very difficult to adjust the pressure to the exact degree required. The danger of permanently arresting conduction, which is very great in mixed nerves, is by no means to be overlooked, since it is well known that by compression motor nerves are much more easily incapacitated for performing their function than sensory ones. Moreover, the pain recurs as soon as the compressing force is removed, and a permanent cure cannot be anticipated; at most it can only shorten the attack. In most instances it is a matter of great difficulty to apply sufficiently strong pressure, and to make it act vertically upon the nerve. It can be accomplished no doubt in certain cases where the nerves of the extremities are affected, but certainly not in the majority of cases of disease of the fifth nerve, because the focus of the affection is, as a rule, situated at a higher point than that at which compression can be applied. Nevertheless, in cases of facial neuralgia, strong pressure is often instinctively made by the patient to relieve the pain, and with more or less advantage. Most authors believe that the relief is effected by interrupting the conduction in the nerves; but, in our opinion, it is rather due to the strong counter-stimulus produced by the pressure, resembling that produced by the violent rubbing of the face, which also causes some alleviation of the pain. Compression is, then, a very inapplicable and in most cases ineffective means of treatment.

b. *By surgical interference*, and, above all, by section of the nerve—*neurotomy*—or by a resection of a longer or shorter segment of the nerve—*neurectomy*. These operations—at least this is the rule—are only applicable in the case of purely sensory nerves, since, if they are performed upon mixed nerves, paralyses result, which in all cases recover very slowly, and sometimes not at all. Such operations on mixed nerves should therefore only be resorted to under the pressure of the most extreme need.

The object of these operations is to inhibit the propagation of the pathological irritation of the nerves to the brain, and by so doing to abolish the pain. They are consequently clearly indicated only in cases of pure peripheral neuralgia, in which a portion of the nerve lying between the seat of the neuralgia and the brain is accessible to the knife. The primary object of the operation is to abolish the pain, and secondly, it is done in the hope that during the period of regeneration and re-establishment of the conduction, by the union of the divided ends of the nerve, a cure of the peripheral focus of disease may be established, a hope that is based on the well-known peripheric degeneration and regeneration that take place in nerves after their division. The hope, however, not unfrequently proves deceptive. However brilliant the immediate results of the operation may be, it is only too often seen that with the restoration of sensibility and the union of the two ends of the nerves, the neuralgia recurs, and sometimes reaches its former intensity, and this naturally occurs more quickly after neurotomy than after neurectomy, a fact which may guide us in deciding which operation to perform. Where a relapse occurs, there is no objection to a repetition of the operation, that is, to the excision of the cicatrix; for, if accomplished with facility, it is a proceeding to which the patient willingly accedes, to obtain complete relief for many months from his tormenting pains.[1]

But good and even brilliant results—however difficult it may be to understand them—occur as a result of neurectomy in cases where the neuralgia is of purely centric origin. The very numerous observations made in this particular branch of surgery by surgeons of great eminence, as by Nussbaum, Schuh, Patruban, Wagner, Nélaton, Gross, Podratzki, etc., have established beyond a doubt that neuralgiæ of centric origin can be temporarily cured by operation, and Bell long ago observed that the operative procedure sometimes produced such an alteration in the tone of the nervous system that a cure of the neuralgia resulted. The conviction is thus forced upon the mind that the strong peripheric stimulus of the operation is the cause—though in what mode it acts is not very obvious—of the disappearance of the neuralgia, and that, like strong counter-stimulation of the skin, it acts as an energetic nervous alterant, and thus leads to at least temporary recovery. The recovery that has sometimes been observed in cases of neuralgia after resection of more or less remote and apparently altogether dissociated nerves, may be explained on the same view (see those reported by Nussbaum and Bardeleben).

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[1] It is conceivable that in neuralgia of centric origin the several attacks are induced by the application of slight sensory stimuli to the periphery. After section of the peripheric nerves these stimuli are no longer conducted centripetally, and the immediate causes of the paroxysms are removed. These either then entirely cease or become less frequent, till peripheral excitations are again transmitted as a consequence of the regeneration of the nerve tissue. In these cases neurectomy would therefore constitute a good palliative. Another cause of relapse lies, without doubt, in the fact that those pathological processes in the nerves, for which the operation was originally performed, recur in the central segment, and thus reproduce the former symptoms.

In the majority of these cases, however, the pain, sooner or later, recurs, thus necessitating repetition of the operation. Higher and still higher points of the nerve, often by very difficult and dangerous operations, as by resection of the upper jaw, etc., are sought to be reached, and not always with permanent results. If it is borne in mind that in most instances the centripetal action of the resection appears to be the really effective part of the proceeding, such large and difficult operations appear unjustifiable, and it is probable that repeated sections of the nerve at the same point would fulfil the same purpose. It is only when good evidence exists that the affection really has a peripheral seat that such severe operations should be performed.

The branches of the fifth nerve are almost the only nerves upon which these severe operations are performed; though the purely sensory cutaneous nerve branches of the extremities may, as a matter of course, be operated upon in this manner. Such operations should not be undertaken upon the mixed nerves of the extremities without due consideration. In some cases, however, as especially in cases of extirpation of neuromata and cicatrices, resection cannot be avoided; the greatest possible attention should then be paid to secure perfect adaptation and reunion of the opposite extremities of the nerve.

If we consider that in many cases extremely difficult and extensive operations are undertaken to attain the desired end, and that in some instances fatal results have occurred; and if we bear in mind the extremely instructive case reported by Wiesner,[1] in which, after repeated severe but futile operations, the application of the galvanic current effected the cure of a severe case of tic douloureux, we cannot avoid arriving at the conclusion that the serious procedure of neurectomy is *only called*

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[1] Berliner klin. Wochenschrift, 1868.

for in cases of very severe and agonizing neuralgia, which have resisted all other means of treatment. In such cases, no doubt, the operation must constitute the ultimate refuge of the patient. The slighter operation of neurotomy, which can be performed subcutaneously, may be more lightly undertaken. Moreover, in neuralgia occurring in stumps after amputation, resection, performed high up, is justifiable, providing the irritative process in the stump has not been propagated to too great a distance upwards (Mitchell).

Amputation of a limb and resection of a joint are procedures which should be stricken off the list of remedies for neuralgia ; it is amazing that they should ever have been undertaken for this purpose. These operations are no more effective than simple section of the nerve trunks implicated, and as such operations are not unattended with danger to life, they cannot reasonably be performed either in peripheral or central neuralgiæ, unless indeed some other disease exist, in addition to the neuralgia, which calls for their performance.

Here, lastly, is the place to add a few words upon the *arrest of the arterial blood supply, by operative means*, as a remedy for neuralgia. Trousseau endeavored to effect this by section and subsequent compression of the arteries, and obtained brilliant results from section of the temporal and occipital arteries in cases of neuralgia of the head. This small operation might very well be more frequently done. Nussbaum and Patruban have also recently tied the carotid artery of the affected side for severe facial neuralgia, and have effected a radical cure of the disease. Such a severe operation as this, and one so dangerous to life, should, of course, only be undertaken in the most desperate cases.

Nussbaum[1] has recently introduced another operative proceeding for the cure of neuroses, which, however, has not been sufficiently frequently repeated to permit any positive statements to be made in regard to its value. It consists in the exposure and extension of the peripheric nerves implicated in the diseased processes. Nussbaum removed by this means a painful contracture, with anæsthesia, in the left leg; Gaertner, likewise, an old painful paralytic muscular contracture of the right arm; and Patruban[1] cured by the same means a severe and obstinate case of sciatica. The true value of such bold proceedings must be determined by further experience.

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[1] *Nussbaum*, Deutsche Zeitschrift f. Chirurgie, Bd. I., 1874.

After this enumeration of the means at our disposal for the treatment of neuralgia, a *few general observations may be made upon the plan to be adopted in particular cases.* These remarks, however, it is obvious, must be very general in their character.

In every case a careful examination should be undertaken to discover the cause of the disease, in order that the treatment may be primarily directed to its removal. The patient desires to be freed from his pain as quickly as possible, and hence it is requisite to apply one of the more certain palliative means, the rather as the more important of these usually correspond to the *indicatio morbi;* electricity or the subcutaneous injection of morphia may therefore be at once applied, or a combination of morphia and quinine prescribed internally. Several of these remedies may be employed coincidently, as galvanization and injections. Additional means in properly selected cases are found in derivatives, as especially flying blisters, irritating applications to the skin, veratria ointment, and the like, or in cold, or in the use of baths and dietetic treatment, which must be modified to suit the circumstances of the individual case. Where no direct or immediate cause can be discovered, and where palliatives really only act as such, it becomes necessary to pass from the continuous use of them to the proper specifics, and arsenic, zinc, bromide of potassium, turpentine, etc., should be prescribed in accordance with the indications for the one or the other.

The remedies selected should of course be employed persistently; but too much stress must not be laid upon this in the case of the narcotics, on account of the unpleasant symptoms

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[1] *Patruban*, Allgem. Wien. med. Zeit., 1872, No. 43 et seq.

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which are apt to follow the withdrawal of these remedies after they have been used for a long period. If the ordinary or more reliable remedies are found to be ineffective, recourse must be had to other specifics, and we may endeavor to produce a favorable action on the whole organism, and especially upon the nervous system, by the energetic employment of baths, carefully regulated diet, cold-water cure, etc. In each case the particular conditions must determine the proceedings to be adopted. Surgical interference is the last resource in the worst or most obstinate cases.

The choice of means for alleviating the neuralgic paroxysms must be left to the tact of the physician. The following means are at his disposal: absolute rest, warmth, warm hand- and foot-baths, the slighter derivatives (sinapisms, friction, the use of the flesh brush), pressure, attention to the bowels, the rapid production of sweating by enveloping the patient in a damp sheet and then covering him with woollen wraps, the cautious administration of chloroform, and the careful hypodermic injection of morphia, which is a remedy of sovereign value, and one that is most to be relied upon in severe cases.

Further details, with some modifications of the general treatment of neuralgia above laid down, will be found in the descriptions of the several forms of neuralgia.

2. Neuralgia of the Individual Nerves.

a. *Neuralgia of the Fifth Nerve.—Neuralgia Quinti.—Prosopalgia.—Fothergill's Facial Pain.—Tic Douloureux.*

J. Fothergill, Med. Observations and Inquiries, Vol. V., 1773.—*Pujol*, Essai sur le tic doulour. Paris, 1787.—*Méglin*, Recherch. sur la névr. faciale. Strassb., 1816.—*Masius*, Beiträge zu einer künft. Monographie des Gesichtsschmerzes. Hecker's liter. Annal., 1826.—*John Scott*, Cases of Tic Doul. and other Forms of Neuralgia. Lond., 1834.—*Schauer*, Gesichtsschm. als Symptom, Casper's Wochensch., 1838.—*Romberg*, Neuralg. nervi V. specimen. Berol., 1840—*K. H. W. Barth*, Mehrjähr. sorgfält. Beobachtung über den Gesichtsschm. Halle, 1825.—*Notta*, Arch. génér., 1854.—*Wiesner*, Berl. klin. Woch., 1868, Nos. 17 and 18.—Compare further the manuals and text-books of *Romberg*, *Hasse*, *Rosenthal*, *Eulenburg*, and others, and the works on electro-therapeutics.—

Valleix, l. c., pp. 7–184; *Anstie*, l. c.; *Trousseau*, Névralg. épileptiforme, Arch. génér., 1853.

Schuh, Ueber Gesichtsneuralg. u. Nervenresection, 1858.—*v. Bruns*, Durchschneidung der Gesichtsnerven bei Gesichtsschmerz. Tübing., 1859.—*Bratsch*, Bericht über 93 Nervenresectionen, 5 Carotisunterbindungen, etc. von Prof. *Nussbaum* ausgeführt. Bayr. ärzt. Intell., 1863.—*A. Wagner*, Ueb. nervös. Gesichtsschm. und seine Behandl. durch Neurectomie, Arch. f. klin. Chir., XI., 1869.—*Patruban*, Unterb. d. Carotis comm., Woch. d. Gesellsch. d. Wien. Aerzte, 1866.

General Account of the Disease.—Under the term *trigeminal neuralgia*, or *prosopalgia*, all those forms of neuralgia are included which implicate the fifth pair of nerves as a whole, or its several sensory branches in particular, and which may therefore affect the various regions to which it is distributed. The whole area supplied by it may be the seat of the neuralgia, though this, in the strictest sense of the word, cannot easily occur; or particular branches may only be affected, of which some, as the supra- and infraorbital and the mental branches, are more frequently affected; whilst others, as the lingual, anterior auricular, and recurrent nerves (of the dura mater) are rarely or never affected. With the anatomical knowledge of the present day, it need scarcely be said that the facial nerve can never be the seat of neuralgia, its frequent apparent participation being due exclusively to the intermixture of the sensory fibres of the fifth (rami communicantes facialis).

Prosopalgia is one of the forms of neuralgia that is most frequently met with. It is of daily occurrence in all possible forms, of which fortunately the most severe are the least common. It is almost invariably limited to one side, though many instances of its affecting both sides have been recorded. It does not appear that one side of the face is more liable to it than the other.

Etiology and Pathogenesis.

A sufficient explanation of the frequent occurrence of prosopalgia is afforded by the extensive distribution and subdivision of the fifth nerve, by the anatomical relations of its branches, which have to traverse numerous fissures, foramina, and canals in bones, and are consequently exposed to numerous mechanical injuries; by the relations of its branches to various important organs, the disturbances of which may extend to the nerves supplying them; and, lastly, by the circumstance that the face is more exposed than other parts of the body to external injuries, cold, wounds, etc. In regard to the essential nature of prosopalgia and its pathogenesis, nothing further can be added to what has already been said in treating of neuralgia in the general sense of the term; and only a few of the causes may here be briefly mentioned.

Amongst the *predisposing causes heredity* occupies a foremost place, and many instances have been recorded in which the disease has affected several generations of a family consecutively. Still more important is the existence of a *neuralgic diathesis*, whether inherited or congenital, which very often displays itself by the occurrence of facial pain, and every practitioner is familiar with the fact that prosopalgia occurs with great frequency in so-called nervous persons, and in those who are hysterical and inclined to various nervous disorders. In regard to *age*, statistics demonstrate that it is very rare in childhood (Chaponnière met with only two children under ten years of age in 119 cases), that it is much more frequent in middle life, and that it is very common in moderately advanced age. The commencement of the worst and most incurable cases is often coincident with commencing old age. *Sex* appears to exert a distinct influence, the accounts given by most authors showing it to be more frequent in women than in men.[1] The occurrence of the disease here, also, as in other forms of neuralgia, has a certain relation to the period of sexual activity. In women the first outbreak of prosopalgia often takes place at the grand climacteric. All sexual excesses or over-stimulation tend to favor the predisposition to the disease. Anæmia, general disturbances of nutrition, dyscrasiæ, etc., play here the same part as in all neuralgiæ. The advent of senescence, with fatty degeneration of the tissues and diseases of the arteries, seems to be of special importance in the origination of this affection. The predisposing influence of changes of season and temperature has not as yet been sufficiently investigated.

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[1] According to *Valleix* the proportion of women to men is 143 : 124; *Hasse*, 30 : 27; *Schramm*, 136 : 59; *Erb*, 51 : 21.

Amongst the *occasional causes* of prosopalgia, *malarial infection* is to be particularly noticed. It produces typical forms of the affection, which are almost exclusively localized in the ramus supraorbitalis, and are further characterized by their endemic or epidemic occurrence in malarial districts, their regularly periodic course (quotidian, tertian, or other, though rarely higher, types), and their easy curability by quinine. The next most frequent cause is *catching cold*, which is easily explained by the exposed position that most of the branches of the trigeminal nerves occupy. There can be little doubt that a large proportion both of the slighter and the more transient cases, and of the obstinate chronic forms of facialgia, are due to catching cold in one form or another, whether from exposure to cold draughts of air, or from wet clothes, damp feet, etc., though the exact mode in which such causes produce their effects is still obscure.

Wounds and foreign bodies are seldom causes of neuralgia of the face. A number of cases, however, are on record in which the disease has been brought on by injury, or where a foreign body, such as a spiculum of glass, fragment of pottery, piece of lead, etc., has been discovered on examination, and the removal of which has effected the cure of the neuralgia.

Diseases of adjoining organs constitute a very frequent cause of neuralgia of the face, and especially of its most severe forms. Diseases of the cranial and facial bones deserve particular mention in this respect, on account of the intimate relations that they bear to the branches of the fifth nerve. Thickenings and hyperostoses of these bones, inflammatory enlargements of them and periostitis, contraction of the different bony canals, caries, sequestra, and exostoses, especially of the teeth and their roots, have all been demonstrated to be causes.

In the next place, neoplastic formations of the most diverse kinds and situated in the most various places are apt to produce prosopalgia, if they mechanically injure the trunk or branches of the fifth, or excite more active disease in them by contiguity. Such effects are particularly likely to occur in the case of tumors at the base of the skull or in the dura mater, and of the jaws. Lastly, the well-known case reported by Romberg shows that *an aneurysmal dilatation of the internal carotid artery* near the Gasserian ganglion is a possible cause of prosopalgia, and this is especially to be borne in mind where indications of atheroma and of aneurismal dilatation are clearly present in the trunk of the carotid and in its principal branches.

Anatomical changes in the nerves and in the brain have not unfrequently been observed, partly in post-mortem examinations, and partly in portions of nerves removed by resection. The reports, however, of the pathological conditions observed, do not agree. Thus, for example, in the nerves themselves (trunk or branches), the neurilemma has been found by some (Carnochan, Wagner, Podrazky, and others), to be thickened, swollen, and reddened; Laveran and Podrazky report thickening and connective-tissue degeneration of the Gasserian ganglion and of the nerve trunks; Romberg found flattening and atrophy of the ganglion and of its branches; while Allan, Norman, and Sharp found small calcareous and inorganic deposits and concretions in the nerve sheath. Notwithstanding these observations, it must not be passed over without remark, that in a whole series of cases the most minute examination of excised portions of nerves has failed to discover any morbid conditions, and that numerous necropsies have only furnished negative results. It may here be stated that it is probable, on clinical grounds, that both *active and passive hyperæmia* of the nerves may become occasionally causes of prosopalgia, though it is difficult to demonstrate it anatomically. But little evidence has as yet been obtained that *diseases of the brain proper* can cause prosopalgia, simply by affection of the intracerebral fibres, apart from any implication of the intracranial part of the nerve. A few cases are on record where tumors, centres of softening, abscesses, etc., in various parts of the brain have existed concurrently with neuralgia of the fifth nerve, but these are upon the whole very rare, and those persons who are so ready to ascribe an excentric or even a central origin to a large portion of the cases of prosopalgia, would do well to remember this fact.

An important and common cause of neuralgia of the face exists in the various *irritations of peripheral nerves.* The irritation may, in the first place, affect the branches of the fifth nerve, and may be due to the presence of foreign bodies, such as have been already mentioned above. In the next place, irritation of carious teeth may often, though certainly not always, be considered as a cause. The great frequency of caries of the teeth makes it conceivable that it may be quite an independent affection—in those cases in which facialgia is also present—and have no relation to that disease. Retarded appearance of the wisdom teeth, and false development of the same, may be causes of neuralgia of the face. Then, again, diseases of the nasal and frontal sinuses may constitute causes of neuralgia of the face, and it is very frequently caused by irritation of the eyes, such as may be produced by over-exerting them, and by reading in a bad or too brilliant light, especially if there happen to be a natural predisposition to disease in them. How far this is true with regard to those diseases of the eyes that are not unfrequently complicated with neuralgia, as glaucoma, herpes of the conjunctiva, etc., and whether these are not, in some cases, sequelæ and concomitant phenomena of the neuralgia, is still not yet satisfactorily determined, and will again be noticed further on.

But trigeminal neuralgia may also proceed from irritation of remote nerve territories. Anstie observed two cases, in which injuries, in one case of the ulnar, in the other, of the occipital nerve, caused neuralgia of the face, and in which the etiological connection between the injury and the nenralgia was, in both cases, pretty clearly demonstrable. To this category also belong the cases in which the prosopalgia is referrible to intestinal irritation (as when worms are present, and there is well-marked constipation), and those in which irritation of the generative organs exists, whether owing to functional over-excitation or to uterine or ovarian disease, etc.

Lastly, according to Anstie, *excessive psychical activity*, whether owing to overwork or to care and anxiety, may be regarded as a direct course of prosopalgia.

In what relations these conditions stand to neuralgia, and what is the mechanism of their action, we have only presumptive evidence; and it is, therefore, still allowable to employ the meaningless title of "reflex neuralgia" to distinguish a certain number of these cases.

For the sake of completeness we shall include gout, rheumatism, hæmorrhoids, and suppression of the perspiration of the feet as possible, though not certainly ascertained, causes of prosopalgia.

Symptomatology.

The principal features of prosopalgia are typical of those of neuralgic affections generally, and, in fact, it constitutes the model from which the classical descriptions of neuralgic diseases and their paroxysms are for the most part drawn. Its essential characters have, therefore, been already descrbed in the preceding section. It presents, however, some modifications, partly owing to the anatomical distribution of the nerve and the implication of certain twigs and branches, and partly to certain physiological relations of this particular nerve.

As *prodromata* of the disease it is common to find the patients complaining of feelings of pressure and tension, of stiffness, itching, and formication, lasting for hours or days upon that side of the face which is threatened with the attack; whilst in other instances there are flying pains in the teeth, slight general malaise, shivering, and the like. In rare cases an actual aura is perceived proceeding from different parts of the body, and regularly ushering in the several paroxysms. Sometimes, however, the attacks come on suddenly, and without any premonitory symptoms.

During the *paroxysm* the pain usually increases in intensity to such a pitch as to beggar description. The severest degrees of pain that can be experienced are felt, and the patient is incapable of finding words that will describe the depth of the misery he is enduring. In the slighter cases moderate pain only is felt, which is in a few instances continuous throughout the whole region affected, the steady unintermitting pain being interrupted now and again by lightning-like strokes; but in most cases the attacks consist of a succession of tearing and boring pains, felt now in this region and now in that. The worst cases are characterized by the occurrence of lancinating and lightning-like pains of the most severe nature, radiating through the patient with fearful vehemence, coming on sometimes with, and sometimes without, obvious cause, and leading him to use the most hyperbolical expressions in regard to the pain, and, as though driven to desperation, he makes the most extraordinary grimaces, and adopts all kinds of expedients to relieve the pain. After a few moments the pain vanishes, often only to reappear with similar characters in some other place. These lancinating pains are frequently located in the bones, and the patient is unable to express their intensity in sufficiently vivid terms. He states that it is as if he were struck by lightning, as if a knife were run into him, or he were penetrated by a red-hot wire, or as if all the bones of his face were being torn asunder. Coincidently with this, various vaso-motor and motorial phenomena accompany the disease in the face, and will be immediately described. The patient resorts to all kinds of expedients to mitigate his sufferings; one, for example, will press a handkerchief firmly against the affected half of the face, or will compress certain painful points with all his might; and another will rub the skin of his face until he rubs it off, and makes a sore place; whilst yet another will set or grind his teeth, or press his head firmly against some hard body. Speech is temporarily suppressed. The face assumes the expression of the most intense suffering and despair, and the patient is for a little while entirely subjugated by the pain. In most instances the paroxysms are short, and leave behind either no further inconvenience beyond exhaustion, or only a dull feeling of pain, which, as compared with the pain of the paroxysm, is easily borne.

The frequency with which the paroxysms recur varies at different times, and with the particular case. They increase or diminish in frequency with the changes of the seasons of the year, and their recurrence is associated with different conditions of the bodily health, and is affected by all circumstances that exert a depressing influence upon, or that improve the general

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health. In many cases, and at certain times, the slightest action of some external cause is sufficient to bring on an attack, and the patient often encounters such influences almost with terror, because he knows they are certain to induce a paroxysm. Thus it may be observed that a slight draught of air, touching the face in washing or shaving, speaking, chewing, sneezing, a bright light or loud sound, any mental excitation, the entrance of a stranger or of the physician into the room, induces an attack, and it becomes obvious that the patient's general health is suffering, and that life itself is almost insupportable on such terms.

During the attacks, in almost all instances, and occasionally in the periods of intermission, *painful points* may be demonstrated at various places in the course of the branches of the fifth, the position of which in the several forms will be hereafter more exactly given. It may, however, be remarked that the *point apophysaire*, so strongly dwelt upon by Trousseau, may also be discovered in many cases of facial neuralgia, as a point extremely sensitive to pressure over the spinous processes of the second and third cervical vertebræ, or over the external occipital protuberance.

In no other form of neuralgia are the concomitant nervous symptoms so numerous and diverse as in this, which results from the intimate connections that exist between the fifth and so many other nerves of the body.

Amongst the *concomitant sensory phenomena* the radiation of pain plays an important part. But few cases are in fact met with in which, at the height of the paroxysm, an extension of pain does not take place, affecting either the branches of the fifth nerve itself, or the occipital nerves, or those of some other more remote nerve region. Irradiated pain is thus sometimes felt in the neck and shoulders, in the mammary gland, in the intercostal nerves, and even in the extremities.

In the affected part there is also not unfrequently a feeling of formication, of slight numbness and furriness. This may even extend, as I have myself seen in one case, to the teeth, gums, and corresponding half of the tongue. Such a condition is often accompanied with obvious changes in the sensibility of the skin (anæsthesia or hyperæsthesia), and the rule laid down by Nothnagel holds good, that hyperæsthesia for the most part occurs in recent, anæsthesia chiefly in chronic, cases of neuralgia of the face. I have, however, met with well-marked anæsthesia in quite recent cases, and I have occasionally, though rarely, observed an entire absence of all demonstrable disturbance of sensibility.

A high degree of anæsthesia occurs frequently in serious organic disease of the fifth nerve coincidently with neuralgia of it (anæsthesia dolorosa), and has then a far deeper significance.

In a few very rare cases *disturbances of the organs of special sense* have been observed. Notta mentions one or two cases of amblyopia and amaurosis which seemed to be referrible to the neuralgia of the face, and which disappeared with its cessation. Disorders of the sense of hearing, occurring as concomitant symptoms of prosopalgia, are somewhat more doubtful, although the innervation of the musculus tensor tympani by the branches of the fifth nerve have rendered it an event of possible occurrence. Disturbances of the gustatory sense, which might result from the implication of the chorda tympani, are seldom mentioned. Photophobia, however, must not be omitted as a not unfrequent phenomenon of neuralgia of the upper branch of the nerve.

Amongst the *concomitant motor phenomena* convulsive contractions of various muscles chiefly attract attention. The most common of these affections are undoubtedly reflex contractions in the muscles supplied by the facial nerve, sometimes appearing in the form of blepharospasm, sometimes as simple contractions of the muscles at the angle of the mouth, at others as a distortion of the muscles of the face, or lastly as true convulsive tic. A case considered to be one of facial spasm was recently sent to me, which, on closer investigation, proved to be one of tic douloureux. Spasmodic contraction of the muscles of mastication is of rare occurrence, yet both tonic and clonic spasms have been observed in them. These must not be mistaken for the energetic voluntary contractions that are made with the object of rendering the jaws immovable or that simply constitute an expression of pain. Paralysis of the muscles of mastication very rarely occurs, and only in cases where some organic lesion implicates the motor as well as the sensory portion of the fifth. Spasmodic movements of the tongue, such as continual rolling of it in the mouth, occurring in very severe cases, are to be regarded as reflex phenomena.

These spasmodic irritations rarely extend to the muscles of the body generally, yet Sinclair Holden has recorded a case in which general tonic convulsions occurred during the attacks. In hysterical patients, the neuralgic paroxysms are frequently the starting-points of general hysterical convulsions.

As regards the paralytic phenomena described here and there as occurring in the muscles of the eye (mydriasis, ptosis, and diverging strabismus),[1] and their relation to neuralgia, no positive statements can be made in the present state of our knowledge, but it is probable that they are only accidental complications, perhaps only co-effects of the same cause.

Vaso-motor disturbances are of very common occurrence. The abnormal pallor of the face, which is often present for a short time at the commencement of the attack, is usually replaced by a degree of redness that may become very remarkable, the face assuming an intensely ruddy, and often bright or polished appearance, with slight doughy swelling. The redness extends over such portions of the mucous membranes as are visible, and it especially affects the conjunctiva, which often presents a high degree of hyperæmia, that may even rise to chemosis. Hyperæmia of the nasal cavity of the affected side and of the corresponding half of the buccal mucous membrane has been observed. Visible perspiration of the affected half of the face has also been noticed in a few cases. Lastly, strong pulsation of the carotids, and also of the facial and temporal arteries, may be observed during the attack.

Physiology enables us to explain why *secretory disturbances* are so common in neuralgia of the fifth nerve, since it has shown the direct or indirect dependence of various secretions upon the functional activity of the fifth nerve. By far the most frequent of these disturbances is *augmented lachrymal secretion*. This, with the marked hyperæmia of the conjunctiva, is one of the most common phenomena accompanying neuralgia of the first and second divisions of the nerve, and admits of very easy explanation, since experimental researches have shown that both the lachrymal and orbital nerves contain secretory nerves for the lachrymal gland, irritation of which causes a flow of tears (Herzenstein, Wolferz), and that irritation of the sensory branches of the first and second divisions of the fifth also increases by reflex excitation the secretion of tears. Increased salivary secretion is somewhat less frequently observed ; the patients describe it as a rapid accumulation of a saltish fluid in the mouth. This symptom is easily explained by the fact that the salivary secretion can be increased, through reflex action, by irritation of the fifth nerve. On the other hand, it is still undetermined whether the secretory filaments (Rami linguales), which run from the third branch of the fifth to the submaxillary and sublingual glands, belong originally to this branch or to the chorda tympani. Still more rarely *an augmented secretion from the nasal mucous membrane* is observed. This is of a watery, mucous character (rarely mingled with blood), and should not be confounded with the discharge from the nose, which is due to the excessive secretion of tears. The researches of Vulpian, showing that irritation of one spheno-palatine ganglion causes increased secretion of the nasal mucous membrane of the same side, afford a sufficient explanation of this pathological phenomenon.

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[1] See cases described by *Notta*.

The *trophic disturbances*, already alluded to in the general account of neuralgia, are most frequently found in just these very cases of prosopalgia. Amongst these are swelling and hypertrophy of the face, partly owing to œdema or actual hypertrophy of the skin, partly to the abnormal growth of the subcutaneous fatty tissue, and partly to hypertrophy of the facial bones, and is most frequent in chronic and obstinate cases. The changes in the hair that have been already described, such as roughness and bristling, periodic or persistent whiteness, and falling-off or excessive growth, are most frequently met with, by far, in cases of neuralgia of the face.

In the next place, various inflammatory conditions of the skin and other tissues frequently accompany neuralgia of the face.

This is especially the case with herpetic eruptions in the region of distribution of the affected nerve, which may be collectively included under the term herpes zoster. Zoster ophthalmicus or frontalis, in the region of distribution of the first division, is most common, and the conjunctiva and cornea are not unfrequently implicated; but I have seen herpes labialis in neuralgiæ of the second and third divisions of the nerve. Erysipelas in the region of the nerve affected is of much rarer occurrence, and, if present, usually disappears in the course of a few days. Anstie mentions subacute inflammation of the periosteum and of the fibrous membranes in the neighborhood of the painful points as often causing well-marked swelling. Lastly, the trophic disturbances of the eye, which are occasionally observed in neuralgiæ of the first branch of the fifth, are of great importance, although their relations to neuralgia have not been quite satisfactorily determined. Herpes conjunctivæ has undoubtedly the same significance as zoster of the skin. The neuroparalytic ophthalmia, now and then observed, is also, no doubt, referrible to the trophic fibres of the fifth, the existence of which has been so much doubted by physiologists. As a rule, this affection only occurs when the lesion of the fifth nerve is severe. How far the statements of Anstie, that iritis frequently originates as a consequence of neuralgia, and that glaucoma simplex in certain cases depends upon neuralgia, are correct, must be established by further observations, especially by the ophthalmologist.

Psychical disorders are more common in prosopalgia than in any other form of neuralgia, though it is extraordinary with what good temper many invalids endure their severe sufferings. In the worst cases, however, melancholia, extreme excitability, and weariness with life are liable to be produced, and in some instances the patients have put an end to their troubles by suicide.

Disturbances of the general health are only observed in very severe cases. The dyspepsia and sleeplessness produced by the disease are then apt to cause impairment of nutrition, which ultimately leads to exhaustion and marasmus. In neuralgia resulting from malaria, the successive stages of shivering, heat, and sweating have been observed to accompany the attacks (Romberg).

Varieties of Neuralgia of the Face.—It is not in all cases that the whole of the fifth nerve is affected with neuralgia; far more commonly it has its seat chiefly or exclusively in particular branches. Various forms of it thus admit of being described.

Neuralgia of the First Branch of the Fifth Nerve (*Neuralgia Ophthalmica*) may affect the whole region of distribution of this nerve or that only of some particular branch. In accorddance with this the pain may extend from the forehead to the vertex and temples, into the upper eyelid, to the root of the nose, and downwards as far as the tip, and through the ethmoidal twig even into the anterior part of the nasal cavities. The eye may itself become, through the ciliary nerves, the seat of violent neuralgic pains (ciliary neuralgia). No observations appear to be on record in regard to the participation of the nervus recurrens ophthalmicus (which is distributed to the dura mater and to the tentorium cerebelli) in neuralgia of the first division of the fifth nerve. The several branches of the ophthalmic nerve may all occasionally become affected by neuralgia, but the supraorbital is by far the most liable, and we then have before us the well-known *neuralgia supraorbitalis*, the characteristic features of which are pain in the forehead, spreading into the upper eyelid and to the root of the nose, hyperæmia of the conjunctiva, lachrymation, etc. In almost all instances there is a well-marked painful point at the foramen supraorbitale. This is perhaps the most frequently observed of all painful points, but the nerve is often very tender to the touch throughout its whole course. Less common painful points are met with in the upper eyelid (palpebral point), on the nose, at the point where the ethmoidal nerve emerges from the nasal cartilage (nasal point), at the inner angle of the eye corresponding to the supratrochlear nerve, on the tuberosity of the parietal bone (parietal point); lastly, the eye itself is often very sensitive to the touch. Supraorbital neuralgia is the form which most frequently results from exposure to cold; it is, moreover, the almost exclusive seat of malarial neuralgia. The concomitant phenomena are usually very strongly marked in this form. The course of the disease is, upon the whole, mild and short, and the treatment successful.

Neuralgia of the Second Division of the Fifth Nerve (*Neuralgia Supramaxillaris*) affects the region of distribution of the second branch of the fifth nerve, and the pain may be situated in the cheek, in the eyelid, in the lateral portion of the nose and upper lip (neuralgia of the nervus infraorbitalis), in the zygomatic bone and anterior temporal region (neuralgia of the nervus orbitalis seu subcutaneus malæ), in the upper row of teeth (affecting the nervi alveolares superiores), and lastly, in the nasal cavities and the gums (affecting the sphenopalatine nerve, with its branches). I entertain no doubt that the nervus recurrens supramaxillaris (like the recurrens inframaxillaris of the third division) is occasionally implicated in the neuralgic affection; for a patient told me distinctly that during the attacks she experienced a deep-seated boring pain "quite in the inside of her head." Anstie also mentions, under the term "brain neuralgia," some cases of a similar nature.

Amongst the branches of the second division of the fifth, the sphenopalatine nerve is the one that is most rarely affected with neuralgia; patients seldom complain of pain in the gums or nasal cavities. The orbital nerve (Henle) is rather more liable to be affected with isolated neuralgia, the seat of the pain in this case being easily localized. But the infraorbital nerve is by far the most frequently implicated (*neuralgia infraorbitalis*), and the characteristic pain is localized in the cheek, upper lip, upper row of teeth, and neighborhood of the zygomatic arch. The more severe forms of facial neuralgia, if limited to one branch, are very commonly seated in the infraorbital nerve, which may, perhaps, be explained by the length of its course through a narrow, bony canal.

The principal painful point in this form of neuralgia is the infraorbital point over the infraorbital foramen; the next one is the malar point, at the point of emergence of the nervus subcutaneus malæ; and less frequent painful points have been demonstrated in the upper lip (labial point), in the gums, or at various points of the alveolar process of the upper jaw. During

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the attacks they are almost always present, but only occasionally during the intermissions.

Dr. Gross[1] describes a special form of neuralgia, which has its seat in the toothless remains of the alveolar processes, especially of the upper jaw, of old people. It presents no features distinguishing it from the ordinary forms of tic douloureux, except the locality. Gross entertains no doubt that the more abundant deposit of osseous tissue in the toothless alveolar processes compresses and irritates the nerves traversing them, and thus occasions the neuralgia. In accordance with this, he states that he has in many instances effected a permanent cure by resection of the alveolar process.

Neuralgia of the Third Division of the Fifth Nerve (*Neuralgia Inframaxillaris*).—This has its seat in the whole of the extensive region of distribution of the third branch of the fifth pair, and the pain may consequently occur in the region of the lower jaw and of the lower row of teeth, in the chin (nervus alveolaris inferior and nervus mentalis), in the tongue and mucous membrane of the oral cavity (nervus lingualis), in the cheek (nervus buccinatorius and nervus communicans facialis), in the temporal region, anterior part of the auricle of the ear, and external auditory meatus (nervus auriculo-temporalis, nervus meatus auditorii externi, and nervi auriculares anteriores). Upon the whole, isolated neuralgiæ of the third division of the fifth pair are not of frequent occurrence, though there are instances in which the neuralgia extends over the greater part of the above-named branches. The lingualis is the least often involved of all the branches, though it is still sometimes attacked, and numerous instances have been recorded in which it has been the only branch affected (neuralgia lingualis). Neuralgia of the auriculo-temporal branch is more common, either as one of the symptoms of a general neuralgia of the third division of the fifth, or as an isolated neuralgia, instances of which have come under my own observation. In the latter case the localization of the pain in the temporal region and ear is characteristic of the disease. The most frequent seat of the neuralgia is in the nervus alveolaris inferior, in which case the pain affects the lower teeth, the lips and the chin.

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[1] American Journ. of Med. Sciences, July, 1870.

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In accordance with this, the painful point that is most frequently met with is at the foramen mentale; the next most frequent point is in front of the ear, where the auriculo-temporal nerve traverses the zygoma; the other painful points mentioned by Valleix—on the lower lip, at the side of the tongue, and on the alveolar process of the lower jaw—are inconstant and difficult of demonstration. The concomitant symptoms are less marked in this form of neuralgia; the unilateral fur on the tongue, and the increased salivary secretion are noticeable, but special attention should be paid to any disorders of the mobility of the muscles of mastication and of the tongue, because conclusions in regard to the seat and nature of the existing pathological conditions can often be drawn from them.

Any attempt to distinguish between the various forms of neuralgia of the face, from a *clinical* standpoint, can be carried out at the present time only in a very incomplete manner. The attempts of the older physicians to distinguish various forms of "rheumatic," "scrofulous," "hysterical," "organic," "inflammatory," "habitual," "sympathetic," and other neuralgiæ from each other by the characters of the pain and the grouping of the symptoms, must be regarded as practically useless. Moreover, the attempt of Benedict[1] to distinguish two great groups of neuralgiæ of the face (as well as of all other neuralgiæ), differing essentially from each other in their symptoms, seat, prognosis, and treatment, does not at present rest upon a sufficiently scientific basis. In the first group Benedict includes those neuralgiæ the paroxysms of which (both in increasing and diminishing intensity) assume a more continuous type; whilst in his second group he includes those the paroxysms of which are only composed of momentary attacks; this second group, he considers, results from reflex excitation of the vasa nervorum. Until this view, however, has a more satisfactory foundation it cannot be seriously discussed.

After all, the cases which belong to the second group, and for which Benedict especially claims the name of *tic douloureux*, are identical with those that have been rendered classical by Trousseau[1] under the term "epileptiform neuralgia," and which must not here be omitted. Without subscribing to the view of their specific nature maintained by the great French clinicist, it may be admitted that the cases of this kind exhibit great similarity in their characters, and many well-marked features in common, in the clinical phenomena presented, and that they are easily recognizable. These are the cases, for the most part, of extremely obstinate prosopalgia, which last for many years, and are the cause of indescribable suffering to the patients, and which have been the objects of many vivid descriptions of facial pain. Pains of the most violent and lightning-like nature occur, succeeding each other with the greatest rapidity for a few seconds or minutes, and then vanishing with equal suddenness; in many cases they are accompanied by remarkable and violent mimetic reflex convulsions. These paroxysms, which are composed of a succession of momentary attacks, may follow each other in quick succession for a period of hours, days, or even weeks; then follows a respite and the attacks disappear for days, weeks, or years; relapses taking place with almost unfailing certainty. The malady is intensified by everything that debilitates or depresses the general health. The first appearance of the disease usually takes place during advanced age, and then often in its worst form. Such may be considered to be the prominent features of the disease, while its details may be filled in from the preceding account of neuralgia of the face.

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[1] *Benedict*, Anzeig. d. Aerzte in Wien, 1871, No. 5.

This form of neuralgia of the face is, for the most part, regarded as being of centric origin, though the view is not sufficiently supported by the results of post-mortem examinations to be fairly established. In favor of it, to some extent, are the circumstances that this form of neuralgia occurs frequently in families in which there is a decided proclivity to psychoses (Anstie), that it occurs coincidently with epilepsy (Trousseau), and that it is frequently associated with a high degree of nervous excitability and with strongly marked psychical activity (A. Eulenburg).

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[1] *Trousseau*, Archives générales, Janvier, 1853; medic. Klinik, Deutsch v. Culmann, Band II., 1868.

On the other hand, it should be stated that it is frequently limited to special branches of the fifth, and that it passes, without any well-marked line of demarcation, into the slighter and ordinary forms of neuralgia of the face. As long, therefore, as no positive evidence can be obtained in regard to the seat and anatomical causation of the disease, we can only consider the tic douloureux just described as the most severe form of facial neuralgia, which, owing to various circumstances, as either advanced age, tissue degeneration, neuropathic diathesis, or anomalies of constitution, is marked by peculiar intensity and obstinacy. No objection, however, need on this account be raised to the suggestion of Eulenburg, that it should be regarded as a *constitutional* form of neuralgia in opposition to the slighter and more *accidental* forms.

The prognosis of this form is, moreover, not so absolutely bad as Trousseau, who never observed an instance of complete recovery, maintains. With the remedial means of the present day at our disposal it is practicable—and even Trousseau succeeded in accomplishing this—to free the patients, in many cases, for a longer or shorter period, from their pains.

The *course* of neuralgia of the fifth may vary considerably. Sometimes it presents a well-marked periodic character, the paroxysm coming on regularly at certain hours of the day or night, with progressive diminution of the attacks till complete recovery occurs. Much more frequently, however, no periodic character is observable. The attacks take place irregularly, a succession of them occurring at certain times and upon definite occasions; then intervals of variable length, which are sometimes prolonged for weeks, months, and even years. This depends essentially on the causes of the disease, the constitution of the patient, favoring circumstances, etc. The course of the affection is most marked and regular in severe cases of true tic douloureux, in which accurate calculations can be made of the periodic return and increase in the severity of the attacks, and of the subsequent longer or shorter intermission, and it is only in the very worst cases that these intervals become shorter and ultimately disappear altogether.

No definite statement can be made in regard to *the duration* *of the disease.* In some instances it has been known to last for a few hours only,[1] whilst in others it has persisted for thirty years. The more severe cases of tic douloureux continue to affect the patient for the remainder of his life.

The terminations of the disease depend in great part on the nature of the cause, on conditions which are in great measure unknown to us. *Recovery* undoubtedly occurs in the majority of cases, especially in the rheumatic, hysteric, and anæmic forms, and in those arising from malaria. The assumption of a *chronic* or *habitual character* is to be anticipated when there is a well-marked neuropathic predisposition, and when the patient is placed under unfavorable circumstances in regard to his nutrition, or suffers from degeneration of the vessels, or when the disease comes on at an advanced age. The disease is, as a rule, *incurable* when it is a result of severe organic mischief, and in those forms which may be placed in the category of epileptiform neuralgia. *Death* may result either from the primary organic lesion or from the general debility and exhaustion, want of sleep, insufficient food, etc., which the neuralgia produces. More accidental terminations are anæsthesia of the fifth, melancholy and other mental disturbances, cerebral apoplexy, etc.

The diagnosis of trigeminal neuralgia presents no particular difficulties if attention be paid to the general characters of the neuralgiæ. It will thus rest upon the seat and distribution of the pain, its characters and paroxysmal occurrence, the existence of painful points and of the various concomitant symptoms, etc. Mistakes, however, may still be made, and uncertainty exist; now and then it may be confounded with toothache, which last is characterized by its being usually local and continuous, by the tooth or teeth being carious or very sensitive to mechanical shock, or to cold. Toothache, however, it must be remembered, may pass into neuralgia. It may also be mistaken for inflammation of the periosteum of the facial bones, or of the membrane lining the antrum and frontal sinuses, from which again it is distinguished by the kind of pain, its seat, the degree of sensibility to pressure, the local tumefaction, etc. ; for hysterical clavus, which is characterized by being limited to one spot, from which the pain does not radiate, and by the existence of other hysterical manifestations; for headache arising from anæmia or dyspepsia, the peculiarity of the pain being that it has no fixed position, that it is deep-seated, dull, and tensive, and does not increase in paroxysms; for migraine, in which case the pain is deeply seated in the skull, and does not correspond with any special branch of nerve, but is pulsating, and accompanied with vomiting and a high degree of irritability, etc. With a little care and attention no mistake in diagnosis of this kind should occur.

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[1] *Valleix* even records a case which consisted of only a single paroxysm that lasted ten minutes.

The *diagnosis of the seat* of the neuralgia is of great importance, and especially the determination of its peripheral or central seat; but unfortunately the present state of our knowledge enables us to accomplish this only in the minority of cases. Its peripheral seat may be inferred, with some amount of probability, from the limitation of the pain to a definite branch of nerve, from the presence of some evident peripheric cause, from the possibility of cutting short the attack by remedies applied to the periphery, from the presence of painful points during the intervals between the paroxysms, etc. In favor of its central origin are the lancinating character of the pain, and its localization in the bones; the widely distributed reflex contractions; general hyperæsthesia and well-marked mental irritability; the dependence of the pains upon the mimetic movements; absence of painful points in the intervals between the attacks; disturbances in other cerebral nerves. None of these characters can, however, be absolutely relied upon, for the determination of the point in question, but the diagnosis may be made with a certain degree of assurance by giving due weight to all the facts of the case.

These remarks apply with still greater force to the question of diagnosticating the *cause of the disease*—which is a matter of pre-eminent importance. It is unnecessary to repeat here all the points which have an important bearing upon the causes of neuralgia of the face, and which every physician will naturally call to mind in each individual case; it is enough to say that correct conclusions can only be formed on this very important practical point by an extremely careful and complete investigation, and by a calm estimate of all the known facts.

The *prognosis* must be founded essentially on the cause of the disease; where none is discoverable, the prognosis should be given with caution, because even apparently slight cases sometimes prove unexpectedly obstinate, and destroy the hope of recovery by frequent relapses. Malaria, rheumatic conditions, anæmia, caries of the teeth, and other peripheric stimuli, allow of a favorable prognosis being given; and a neuropathic diathesis, advanced age, cachectic conditions of the body, degeneration of the tissues, severe organic diseases, central lesions, etc., lead to an unfavorable prognosis. Habitual, or so-called constitutional neuralgiæ are particularly hopeless; as a rule they only undergo temporary improvement. On the other hand, as a rule, a quick recovery may be anticipated in recent cases coming on with moderate severity, and especially if presenting a periodic character.

Treatment.

The treatment of prosopalgia only too frequently presents extraordinary difficulties, taxing the knowledge and patience of the physician to the utmost, and in spite of all efforts giving an unsatisfactory result. The treatment must be conducted on the broad principles already inculcated in regard to neuralgia generally, and in the remarks that will now be made but few points will be discussed at somewhat greater length, while the majority will only be briefly alluded to.

Not unfrequently the causal indications can be fulfilled. Quinine is highly successful in supraorbital neuralgia, when due to malarial infection; energetic antiphlogistic measures should be adopted in neuritis, osteitis, or periostitis; in cases of rheumatic origin, diaphoresis, rapid derivation, and the internal administration of colchicum and opium should be prescribed. The removal of foreign bodies, cicatrices, tumors, and neuromata, is often attended with brilliant success. Special attention should be paid to the extraction of carious teeth, which, though often useless, is also often attended with good results. This, however, should only be done when the pain obviously proceeds from the affected tooth, when paroxysms can be induced by irritation of it, or when the tooth is very painful on being touched with metal, or with any cold fluid. According to Friedberg the neuralgia is often only alleviated when the damaged, but as yet not painful, tooth is removed. In very excitable and weakly persons the extraction should be performed under the influence of some anæsthetic. It is not allowable to extract sound teeth merely because they ache during the neuralgic paroxysm. In cases of irritation of peripheral organs, or of other nerve regions, the appropriate methods of treatment must be adopted. Particular attention should be paid to the general health, and anæmia, cachexia, and the various dyscrasiæ, as well as the neuropathic predisposition, should be as far as possible improved. This must be done on general principles modified by the circumstances of the particular case. The knowledge of the practical physician here comes into play, and the proper selection of the various tonic, alterative, and other methods of cure, the choice of appropriate baths, cold-water cures, diet, etc., is of the greatest value.

Electricity has recently gained a conspicuous position in the *direct treatment* of neuralgia. Cases are on record in which success has been obtained by its means, although the most severe operative proceedings had been previously adopted without effect (Wiesner); and it may fairly be said that it is essentially owing to the extension of electrical methods of treatment that the prognosis of true tic douloureux is no longer so hopeless as Trousseau represented it to be. Both kinds of electricity are useful. The faradic current cannot well be applied to the face in the form of either the electric brush or the electric moxa, on account of the violent irritation and intense pain which they produce, but they may be applied, as M. Meyer recommends, to the neck; and the suggestion made by Eisenmann may be practised, namely, to endeavor by powerful cutaneous faradization of the external ear to obtain the same results as by cauterization of the helix. Faradization with moist electrodes seems likely to prove more serviceable, the poles being applied to the especially painful points and near the nerve-trunks. In one of my own cases, a faradic current of this kind, passed through the head in front of the ear, with "swelling" currents, after Frommhold's directions, proved more effective even than galvanization. As a general rule, however, treatment with the galvanic current is far preferable to that with the faradic current, and gives much more favorable results. Good results have been obtained from the most various methods of applying the currents. In the treatment of peripheric neuralgiæ of isolated superficial nerves, such as the auriculo-temporal, frontal, inferior alveolar, etc., both electrodes may be placed over the nerve, and a descending stabile[1] current passed through it. The endeavor may be made to obtain a similar action upon the deeper-seated nerves, such as the infra-orbital, lingual, etc., by placing the anode on the neck or behind the ear, and the cathode over the point of emergence of the affected nerve. In the polar method the anode is to be placed upon the specially painful points, and held stationary there, whilst the cathode rests against the back of the neck or on any other indifferent part of the body. In order to exert an action on the main divisions of the nerve at the base of the cranium, and after their emergence through the foramina of the sphenoid bone, it is advantageous to conduct the current transversely through the skull at the appropriate points, the anode being placed on the painful side. In the more severe cases I have found a polar action of the anode at these points, with a pretty strong stabile current, at first increasing and then decreasing in intensity, particularly useful. Benedict recommends that in severe cases galvanic currents should be passed longitudinally and transversely through the skull and along the sympathetic nerve, and I believe I have sometimes seen good results from this method of treatment.

It is the problem of electro-therapeutical research to render indications for the employment of these various methods more precise in the future. At present this is not possible; but it is probable that different methods of galvanization should be applied in the different forms of prosopalgia, according to their causes and seats. But as we are at present, in the majority of cases, ignorant both of the seats and of the causes of the neuralgiæ, we are compelled to employ the several methods in an empirical manner, and especially in the more severe cases to try them one after the other, in order to discover which is the most effective. The results obtained in the slighter and more recent cases, as well as in some of the chronic idiopathic forms, are sometimes very striking. In the more severe cases, though in many instances a certain amount of alleviation may be effected, yet perfect recovery is seldom obtained. But it is just in these cases that we must endeavor by perseverance and the adoption of various modifications in the treatment to attain the wished-for end. Great experience and skill in the technical manipulations of electro-therapeutic means are of course indispensable, and for further details we must refer to treatises on electro-therapeutics.

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[1] The terms "stabile" and "labile" (stabiler und labiler, Strom) have reference, not to the character of the current, but to the manner in which the electrodes are handled. If both electrodes are kept still, the current is stabile; if one or both be made to slide over the skin, the current is labile.—*Translator's Note.*

In regard to the administration of *narcotics*, the principles laid down in speaking of the general methods of treatment in all respects hold good here; but care must be taken not to continue the use of them in too large and active doses, since it is difficult for the patient subsequently to give up their use. It is on this account advantageous to vary the narcotic employed and to prescribe in succession, or alternately, opium, morphia, laudanum, stramonium, hyoscyamus, and belladonna. The chief dependence, however, must always be placed on the opiates, and especially on morphia. Under the pressure of circumstances narcotics have been prescribed in innumerable methods; the most advantageous of these is the hypodermic injection, which, especially in neuralgia of the face, should be used with all possible precaution. The injection should but rarely be made into the skin of the face, because it is very painful, often leaves a mark, and frequently leads to inflammation. The best places are the temporal region, and the side of the neck below the ear. When the pain is well marked and very strictly localized, the injection may, however, be made into the forehead or

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skin of the cheek. Such injections often effect a cure in slighter cases; but for the more severe cases they only constitute a palliative means that in general gradually loses its efficacy, and requires to be administered in doses that are injurious to the system, and even then fail to produce the desired effect. The injection of other narcotics is not followed by any special advantage, though perhaps atropine may be tried, which is recommended by Anstie in the supraorbital neuralgiæ accompanying glaucoma.

The internal administration of narcotics is not very advisable, though there are many cases in which it cannot be dispensed with. The plan adopted by Trousseau, and regarded by him as the only effective palliative treatment of epileptiform neuralgia, namely, by incredibly large doses of opiates, deserves particular mention. Trousseau begins with three grains of morphia, opium, or extract of opium per diem, and rapidly increases the dose, if no unpleasant symptoms are produced (and these rarely occur in this affection), until the pain is alleviated—in some cases to as much as a drachm of morphia (!), or two or three drachms of opium in the course of the day (!). He reports the case of a patient who took daily from four to five drachms of opium, and yet suffered from none of the toxic effects of the drug, whilst he experienced very decided relief from his tormenting pain. By this plan of treatment Trousseau at least effects this, that the attacks in such cases become less frequent and violent, and that they may even intermit for some weeks or months. He expressly calls attention to the fact that the opium must be given in *large doses*, and that the limits beyond which we should not go cannot be determined beforehand. As improvement proceeds, intolerance of the remedy becomes established, and the doses must be reduced. Many persons, however, are unable to take the larger doses.

Belladonna, hyoscyamus, stramonium, etc., may likewise be tried in increasing doses, and occasionally prove useful. The introduction of narcotics by the nose, in the form of watery solution or of powders, may also be mentioned, and has been shown to exert a favorable palliative action, especially in supraorbital neuralgia.

In regard to external treatment, ointments containing opium, veratria, and aconitia may be rubbed in, and atropine fomentations, which are easily applied, may be recommended (see section on Treatment of Neuralgia in General).

The anæsthetics employed in the mode already described not unfrequently prove serviceable. In particular the inhalation of chloroform and a few external applications (as a pledget of cotton wool impregnated with chloroform and inserted into the external auditory meatus, or one wetted with chloroform or ether and placed upon the painful spot) are recognized remedies in the treatment of facial pain.

It is very frequently requisite in cases of facial neuralgia to resort to specifics, and the remarks made in the general part are here also applicable. In the milder periodic, or almost periodic cases, even if they have not a malarial origin, quinine is usually effective. I give it freely in combination with morphia in recent cases of idiopathic or rheumatic supraorbital neuralgia, and for the most part with good results. Arsenic, on the other hand, is the sovereign remedy for the more severe cases. In these, when occurring in patients who have a constitutional predisposition to neuralgia, it is often so decidedly useful that it deserves to be tried again and again. The ordinary amount may be given, or even rather more, without fear of unpleasant results, as three, six, eight, or ten drops of Fowler's solution several times a day, or arsenious acid in doses of from one-sixth to one-third of a grain per diem in divided doses. Nothing further need be said of the remaining specifics, zinc (Meglin's pills), strychnia, nitrate of silver, chloride of gold and sodium, etc. Lecointe has observed good effects from the use of chamomile (continued for some time). Pfaff and others praise the internal administration of turpentine, and others recommend corrosive sublimate, carbonate of iron and iodide of potassium.

Little benefit is derived, as a rule, from the employment of *derivatives*. Vesicants are best applied on the neck, or behind the ear. Various irritating and stimulating salves have been applied to the face. The suggestion that the skin, over the painful points, should be cauterized with nitrate of silver, has met with little favor. Painting with tincture of iodine, acupuncture and electropuncture, aquapuncture, painting the gums with diluted water of ammonia, and the like, are energetic modes of stimulation, which have for the most part only a transient effect, and are less active than the electrical brush. The actual cautery in the form of transcurrent cauterization, the most heroic, and at the same time the most effective of these counter-irritants, has likewise been tried.

Cold is but rarely useful; in general the patient is unable to bear its application, and prefers warm coverings and poultices. In some cases, however, of peripheric, and especially of neuritic neuralgia, cold applications prove extremely serviceable. *Pressure* may be tried without fear in cases of trigeminal neuralgia, since the nerves thus pressed upon are purely sensory ones, the arrest of conduction of which is not followed by any ill results. Energetic pressure may, therefore, always be made upon the nerve affected, or upon the painful points, to see whether it affords relief, and in some cases no doubt it will prove to be of value.

Good results can only be expected from *baths* when there is some definite indication for their use. Sea-water bathing, thermal baths, and cold-water cures are often followed by brilliant results, but often, too, prove useless, or even produce an exacerbation of the disease.

Lastly, there is no form of neuralgia in which *operative proceedings* are so frequently indicated, and have been so frequently performed, as in prosopalgia. The discussions raised by surgeons in regard to the value, the indications, and the methods of performing neurotomy and neurectomy, refer almost exclusively to the fifth nerve. It is certain that in many cases of trigeminal neuralgia there are very definite indications for the operation, and that the majority of these operations are relatively free from danger, and, with the improved methods of modern science, have little effect upon the general health; and that the operation is beneficial to the patient himself is shown by numerous examples in which repeated operations have been undergone, and the surgeon even entreated to still further and bolder proceedings. The misery of such patients must have been witnessed to render it intelligible how earnestly they desire that some means, however severe, should be undertaken which promise to give them even transitory relief from their sufferings.

The operation seems to be indicated, *a*, when the neuralgic affection or the paroxysms appear to have a peripheral origin, and cannot be otherwise cured; *b*, when with doubtful, or even with centric origin, the severity of the disease is so great as to justify, after all other means have been tried, a severe operation. The fact that the operation has afforded relief in many cases, which were well known to be of centric origin, has given material support to this indication. It is obvious that the more severe cases of "epileptiform neuralgia" are those which especially fall into this category, and it is in these that the operation has been already frequently performed.

Before any operation is undertaken, it is of course necessary to ascertain as accurately as possible the particular nerves that are affected, and in this point of view it is especially important to investigate accurately the directions in which the pains are radiated, and to ascertain exactly how high up in the course of the nerve the affection is located, since it is obviously of great importance to operate above this point; naturally also the operator will choose in each case the least dangerous, as well as the most certain plan of operation. In cases where the diagnosis is uncertain, and the result of the proposed operation doubtful, it may perhaps be well in the first instance to practise a slight operation (*ex. gr.*, subcutaneous neurotomy), and then, if the pain return, to resort to more severe measures (such as the excision of as long a portion of the nerve trunk as possible, neurectomy). This must, however, be left to the discretion of the surgeon. Full details in regard to all these points, the indications for the performance of the operation, the subsequent phenomena, and the after-treatment will all be found in the excellent article of A. Wagner.[1] We cannot here discuss the details of the various operations. They have become extraordinarily perfected of late years. Resections have been performed upon every conceivable and almost inaccessible nerve—in the depth of the orbit, and in the pterygoid fossa as far back as to the foramen rotundum—and even resections of the upper and lower jaws have not deterred operators from reaching the nerves affected. The infraorbital nerve is the one upon which operations are most frequently performed, but for special details on these points the reader must refer to works on general and operative surgery.

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[1] *A. Wagner*, Archiv f. klin. Chirurgie, Band XI.

The results that have been obtained are, upon the whole, satisfactory. A report upon 135 cases, by A. Wagner, shows the number of total failures to be 9; of deaths, 6; of relapses, after a few days, 1; after the lapse of some months, 32; after the lapse of some years (up to 3), 20; of relapses not reappearing for some months, 18; not for some years, 25. The duration of the result obtained was not given in 24 cases. If it be considered that all these were extremely severe and obstinate, whilst some were doubtful cases, we shall probably agree with Wagner, when he observes that neurectomy in prosopalgia stands on a scientific basis, and is of high practical value.

In regard to the propriety of performing the operation of tying the carotid, recommended and practised by Nussbaum and Patruban, as a means of effecting a radical cure of neuralgia of the fifth pair, we are not at present in a position to make any positive statement. The results that have been already obtained are, however, so favorable, that the operation is well worthy of further trial. As, however, no precise indications can be given as to when it should be done, we can only turn to it as a last resource in cases where the constant pain experienced by the patient justifies an energetic though somewhat dangerous proceeding.

In regard to the appropriate regiminal treatment in facial neuralgia, there is nothing to add to what has been already given in the general part. As the trigeminus, in consequence of its numerous physiological relations, is capable of being excited and influenced in the most various manner, both by physical and psychical influences, attention should be paid to the removal of all circumstances that may call forth or increase the intensity of the paroxysms. In particular, all physical or intellectual over-exertion, and generally everything likely to exert a depressing influence on the constitution should be avoided, and a temporary retirement from business is often requisite. The diet and mode of life should be directed in accordance with the circumstances surrounding each case, and with the hints and recommendations that have been already given in the general part of this work. Great circumspection and steadiness of purpose are necessary in severe cases.

b. *Neuralgia in the Region of Distribution of the Four Upper Cervical Nerves* (*Plexus cervicalis*).—*Cervico-Occipital Neuralgia.*

Bretschneider, loc. cit.—*Valleix*, loc. cit., pp. 185–224, *Eulenburg*, *Hasse*, loc. cit.—*Bérard*, Dict. de méd., Tome XII., 1836.—*Peter*, Névralgies diaphragmatiques, Archives générales de médecine, 1872, Tome XVII.—*Falot*, Névralgie du Phrenicus, Montpellier méd., 1866.

This upon the whole rare neuralgia has its seat in the region to which the sensory fibres of the four upper cervical nerves are distributed. The following nerves may consequently be implicated in the disease: 1. The *nervus occipitalis major*, which emerges from the deeper tissues, just beneath the skin, between the mastoid process and the spine of the second cervical vertebra, and sends branches to the occipital region, and forward as far as the vertex. 2. The *nervus occipitalis minor*, which is distributed over the side of the back part of the head as far as to the ear. 3. The *nervus auricularis magnus*, which sends branches to the concha and skin over the mastoid process and parotid gland. 4. The *nervus subcutaneus colli inferior*, which is distributed over the whole anterior region of the neck, extending in front as far as to the region beneath the chin, and on the side as far as to the cheek. Lastly, 5. *The nervi supraclaviculares*, the branches of which ramify throughout the clavicular region, the upper thoracic region, and the lower part of the neck.

In this form of neuralgia the pain may consequently be widely distributed, affecting the whole posterior region of the head, as far as to the vertex and ear, the skin of the neck generally, as far downward as the clavicle, and as far upward as the cheek (where anastomosis takes place with the subcutaneus colli superior, a branch of the facial nerve). It is the nervus occipitalis major, however, which is most frequently affected (*neuralgia occipitalis*). The occipitalis minor and auricularis magnus are less liable to become affected, and the implication of the subcutaneus colli and of the supraclavicular nerves is but rarely mentioned.

In regard to the etiology of this form of neuralgia little is known. The predisposing causes are the same as in neuralgia generally. It is frequently attributable to hysteria and anæmia.

Among the exciting causes *exposure to cold* is probably one of the most frequent, and the occurrence of the neuralgia is often very positively referred to a cold draught of air upon the neck, to getting wet, and the like. In a few instances the disease appears to have been due to an *injury* of some kind. *Disease of the upper cervical vertebræ* is both a very common and a very serious cause of the affection. The disease of the vertebræ, leading to occipital neuralgia, may consist in rheumatic or syphilitic periostitis, in caries, or in tumors of all kinds (and here I might state, that I have seen a well-marked case consequent upon the development of an osteoma, growing from the upper cervical vertebræ). Diseases of the cervical lymphatic glands, and in one case aneurism of the vertebral artery, have been observed as causes. Lastly, it is not improbable that diseases of the central nervous system (cervical portion of the spinal cord and brain) may occasionally exhibit as one of their symptoms an occipital neuralgia.

Symptoms.—The attack is usually unilateral, but not unfrequently occurs on both sides. The pain may be exceedingly severe, and is commonly described as tearing and boring, and not unfrequently as distinctly lancinating. It usually extends from the side and upper part of the neck upwards to the vertex, and even to the frontal region; more rarely it runs up to and behind the ear, or forwards towards the cheek, or towards the anterior region of the neck, sending out twinges towards the chin and over the lower jaw; or lastly, it may extend downwards over the clavicles, and backwards to the scapular region. The paroxysmal character of the pain is in general well marked, and the paroxysms may become as intense as those of prosopalgia, and may vary considerably in duration. During the intermission the patients are either entirely free from pain, or experience only a dull sensation of weight or tension. The attacks are brought on by movements, and especially by turning the head, by laughing, mastication, sneezing, etc. The fixed manner in which the patient holds his head during the attacks is very characteristic, as he dreads every movement; but when the attacks have passed off he is again able to move his head freely.

Painful points are often demonstrable in this form of neuralgia; most frequently at the point of emergence of the nervus occipitalis major, about halfway between the mastoid process and the spinous processes of the upper cervical vertebræ (occipital point). The track of the nerve itself over the occiput is often painful, as are also a point over the parietal eminence (the parietal point) and the spinous processes of the upper cervical vertebræ; but such points can rarely be demonstrated in the course of the trunks of the remaining nerves that are implicated, nor in the ear, nor over the clavicle.

The pain in this form of neuralgia is rarely limited to any special nerve region, but, on the contrary, radiates widely, most frequently affecting the brows and temples, and the cheeks; various branches of the brachial plexus and of the intercostal nerves become painful, and this leads to this form of neuralgia being mistaken for other forms, and especially for neuralgia of the fifth pair.

The ordinary disturbances of sensibility may now and then be observed; hyperæsthesia of the skin in the occipital region, for example, has been reported, in which the hair was tender to the touch. I have myself observed slight anæsthesia over the whole region of distribution of both occipital nerves and of the auricularis magnus, connected with a subjective sensation of numbness. During the paroxysms, *convulsive phenomena* not unfrequently occur in the cervical muscles, such as cramps and spasms in them, and often a tonic contraction, which, however, is sometimes induced by the patient himself for the purpose of keeping his head in a fixed position. Contractions rarely occur in the facial muscles. Little is known in regard to vaso-motor and trophic disturbances. Anstie mentions enlargement of the lymphatic glands as a consequence of this form of neuralgia. I have observed frequent vomiting and extreme emaciation in a very severe case of the disease, which is otherwise deserving of being briefly reported.

On the 8th of October, 1862, a peasant boy, æt. 15, named Gosswin Heiler, was brought, in an extreme state of emaciation and debility, to the clinic of Prof. Friedreich. The anamnesis, which the boy—reduced to a perfect skeleton—gives with tolerable clearness, indicates a duration of two or three years for the affection, with rough treatment on the part of the step-parents, and bad and insufficient nourishment. The attacks have gradually become more frequent and violent, and the general nutrition of the body, in consequence of the associated vomiting, constantly poorer. The patient complains of extremely violent pain in the neck and occiput, extending into the back, and occurring in paroxysms, which are always accompanied by vomiting. During the attacks there is great sensitiveness of the skin over the second cervical vertebra and also over some of the dorsal vertebræ. Movements of the head made during the attacks cause the most frightful pain, whilst at other times the head can be moved freely without inconvenience. The patient usually lies bent in bed, as if in a state of opisthotonos, and can neither stand nor walk, nor even sit up in bed without assistance. All food is rejected by vomiting. The emaciation and weakness are almost inconceivable. The intellectual faculties are unaffected, and no other anomalies are present. There are no symptoms of disease of the central organs. After the application of cold, narcotic liniments, etc., had been employed without effect, morphia injections were tried in doses of one-twelfth, increasing to one-sixth of a grain, with surprisingly rapid improvement. In the course of a few days the pain had entirely disappeared, the vomiting ceased, and the appetite and the power of locomotion returned. The patient recovered by degrees completely, so that he undertook a place as a messenger. His weight on the 2d December, when considerable improvement had already taken place, was fifty-six pounds, and on the 23d of April it was seventy-four pounds. No recurrence of the neuralgia occurred.

The course of cervico-occipital neuralgia is in general milder than that of prosopalgia, but here also many severe and incurable cases are met with, which resemble epileptiform neuralgia. In both instances this depends on the causes, and any consideration of details would only lead to repetition. The same may be said in regard to the duration and terminations of this form of neuralgia.

The *diagnosis* is usually made with facility, and any confusion of it with rheumatic torticollis (or muscular rheumatism with fixed pain, not rising into paroxysms nor undergoing augmentation, but constantly accompanying certain movements) may easily be avoided. The diagnosis of the causes is both more important and more difficult, and a careful examination of the cervical vertebræ both externally and by way of the pharynx should always be undertaken. The long-recognized symptom, that in diseases of the vertebræ the patient, when raising the head, supports the occiput with the hands, is to be borne in mind.

The prognosis must be deduced from the causes, and from the estimate we may form, on general principles, of all the concomitant conditions.

The *treatment* is in most cases successful; in severe organic diseases, however, palliative treatment is our only resource. In ordinary cases *cutaneous irritants* are commonly prescribed. Flying blisters are often highly successful, as are also the electric brush and the moxa, and transcurrent cauterization; slighter cases may be cured by embrocations with veratria ointment, or a mixture of five parts of turpentine and one of croton oil. *Local depletion* is seldom employed. The action of the galvanic current, applied according to the usual methods, is excellent in recent rheumatic cases, and often also in chronic cases of the same nature. Lastly, an exceedingly good palliative, and not unfrequently curative means, is to be found in the subcutaneous injection of morphia. Specific and general treatment must be based on general principles. In desperate cases, the easily practised resection of the affected nerve trunks may be tried, though it is usually ineffective, since the proper seat of the disease is not accessible.

Under the term *neuralgia diaphragmatica* Peter has recently described a form of neuralgia which has its seat in the phrenic nerve, and is of very frequent occurrence; perhaps, indeed, it is one of the most common neuralgic affections.[1] The phrenic nerve is generally considered to be purely motor, but its mode of origin from several branches of the cervical plexus renders it highly probable that it contains sensory fibres. Peter regards it as a mixed nerve, and anatomists, as Henle, for example,[1] are satisfied that it contains sensory fibres. Physiological evidence on this point has yet to be supplied, however favorable to it the observations and explanations of Peter may appear.

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[1] *Falot* (Montpellier méd., 1866; Virchow and Hirsch Jahresbericht) has already described the same affection, of which he himself had experienced several attacks, under the name of "neuralgia of the phrenic."

Neuralgia of the phrenic (which may be advantageously termed *phrenic neuralgia*, in accordance with the usual terminology of these affections), is characterized by the occurrence of acute pain at the lower and especially the anterior part of the thorax, along the line of attachment of the diaphragm; also by pain in a line corresponding to the course of the phrenic through the chest; lastly, by pain at the point where the nerve arises from the cervical plexus and descends over the scalenus anticus. Pain is also occasionally experienced in the shoulder, neck, and lower jaw, *i.e.*, in the region of the cervical plexus, and radiating into the upper extremity, and especially down the inner side of the upper arm. The pain in the shoulder is so constant and so characteristic, that its occurrence, coincidently with pain at the base of the thorax, immediately arouses the suspicion of phrenic neuralgia. Careful examination demonstrates the existence of a *number of painful points*, namely, 1, the spinous processes of the upper cervical vertebræ, especially from the second to the fifth, and less frequently to the sixth; 2, the phrenic nerve itself, as it pursues its course along the supraclavicular fossa; 3, the line of attachment of the diaphragm, especially anteriorly between the seventh and tenth ribs, and more rarely posteriorly; and lastly, 4, a point over the cartilage of the third rib, the significance of which, however, is not quite obvious.

The pain is more or less continuous, but yet presents exacerbations rising into paroxysms, which are often of a lancinating character, the continuity of the pain being in part perhaps explained by the unceasing movement of the diaphragm. The neuralgia is almost always accompanied by impeded respiration (through impairment of the activity of the diaphragm), and hence exertion, sneezing, coughing, etc., are all rendered difficult and painful. Muscular debility and tremblings are not unfrequently present in the arm. Other concomitant phenomena mentioned by Peter (as palpitation, angina pectoris, and the like) do not belong to phrenic neuralgia as such, but to the lesions complicating or producing it.

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[1] *Henle*, Nervenlehre, p. 472.

Now, although phrenic neuralgia is not unfrequently an independent and primary disease (especially in nervous and anæmic individuals, with whom it is a consequence of exposure to cold, etc.), it is far more commonly a concomitant of some other disease, especially of angina pectoris, and of cardiac and vascular lesions, Basedow's disease, enlargement of the spleen and hepatic disease; and Peter is inclined to refer a part of the ordinary or extraordinary pains occurring in these affections to neuralgic implication of the phrenic, and supports his view by the histories of numerous cases. Phrenic neuralgia especially affects the left side. The diagnosis of this form of neuralgia from other diseases accompanied by similar phenomena, as diaphragmatic pleurisy, pericarditis, gastralgia, pure angina pectoris, etc., does not present extraordinary difficulties, providing careful attention be paid to the particular symptoms in both forms of disease.

The treatment must be that usually pursued in neuralgia. Peter has employed cupping-glasses, vesicants, and subcutaneous injection of morphia. In most cases the thing of chief importance is to treat the fundamental disease.

APPENDIX.

Headache.—Cephalalgia.—Cephalæa.

Bittner, Tract. de cephalalgia. Vienn., 1825.—*Weatherhead*, on Headache, translated by Pfeiffer. Leipzig, 1836.—*Wilkinson King*, on the Seat of Headache, Lond. Med. Gaz., Dec. 27, 1844.—*Patrick J. Murphy*, on Headache and its Varieties, Lancet, 1854. February and March.—*Sieveking*, on Chronic and Periodical Headache, Med. Times and Gazette, 1854, Aug. 12, 19, 26.—*John Add. Symonds*, on Headache, Med. Times, 1858, March-May.—*Lebert*, Handbuch der Allgemeine Pathologie, 1865, p. 528.—*Hasse*, loc. cit., p. 29.—*A. Eulenburg*, Zum Pathologie des Sympathicus, Berlin. klin. Wochensch., 1873, No. 15.—*Benedict*, Nervenpathologie und Electro-Therapie, 1874, p. 185.

Scarcely any symptom of disease of the nervous system is so frequently met with in practice as "headache," and there are few that are susceptible of so many and varied interpretations. Headache occurs more or less constantly, though often only as a deceptive symptom, not only in nervous diseases, but in almost every other form of disease, as in fever, in disturbances of the digestive organs, and in the most diverse inflammations of the head and adjoining parts. It is not intended here to give a complete treatise upon headache, since, as Eisenmann has observed, it would be easy to fill an octavo volume of goodly size with matters pertaining to this subject, but only to make a few brief observations on those forms of headache which in common parlance are ordinarily designated as more or less purely "nervous," and which cannot be included in the ordinary category of neuralgia and the other well-known forms of disease.

And those forms of headache may first be briefly alluded to, which do not properly form the subject of these remarks. The most prominent of these is that caused by *external neuralgiæ* of the sensory nerves. We have already pointed out in our account of trigeminal and occipital neuralgia how this is to be recognized, as, for instance, by the localization of the pain, the painful points, and the concomitant phenomena.

An affection that is frequently included in the category of nervous headaches is *hemicrania* (migraine). Well-marked cases of this neurosis are easily recognized by the paroxysmal periodic occurrence of pain on one side of the head, malaise and vomiting, by hyperæsthesia of the organs of special sense, and by their relation to the catamenia and to errors of diet, etc., for details respecting which the reader is referred to the chapter on hemicrania. Many cases obviously belong to this category, which, however, do not present all the features of a well-marked attack of migraine; as for example, the pains felt by many women on one or both sides of the head, which set in with general feelings of illness, disappear when compression is made on the carotids, persist for variable periods, and not unfrequently alternate with attacks of true migraine. Careful observation, continued for some time, generally enables us to make a correct diagnosis of these cases.

Headache, again, is one of the most constant symptoms in

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fevers, whatever may be the etiology of the fever. In ordinary cases it is moderate in intensity, dull, and deep-seated, situated as it were at the bottom of the orbit or at the base of the skull. It is increased by movements of the head on stooping and the like, and is accompanied by a feeling of light-headedness which not unfrequently is associated with delirium. The diagnosis must be essentially based upon the rise of temperature that accompanies true febrile affections. The precise cause of the headache that accompanies fever has not been very satisfactorily ascertained. Cases no doubt occur in which besides the fever other circumstances concur in producing headache.

Headache is an almost constant symptom in *all inflammatory and ulcerative processes*, and in cases of *neoplastic formations* taking place in the skull and adjoining tissues, and in the *diseases of the higher organs of special sense;* and in these cases it would appear to be caused partly by mechanical and partly by inflammatory irritation of the nerves, and partly also to be due to radiation. To this category belong the headaches that occur in catarrh of the frontal and sphenoidal sinuses ; in inflammatory affections of the skin, of the fascia of the occipito-frontalis, and of the periosteum of the cranial bones ; in syphilitic diseases of the skull ; in caries of the petrous portion of the temporal bone ; in tumors of the base of the cranium ; in lesions of the eye and ear, etc. Such headaches are often held to be of a purely nervous nature if the primary disease develops slowly and without betraying itself by any marked symptom.

Headache, lastly, is a very important symptom in every form of *disease of the brain and of its membranes.* It is a common though certainly not constant concomitant of all such affections. It often becomes very intense, deep-seated, boring, and throbbing. It may be either continuous or paroxysmal in character, and it is increased by movements, by blows on the skull, etc. Its true significance is often first recognized only after other symptoms of cerebral lesion have made their appearance: as delirium, fainting, vomiting, partial paralysis, anæsthesia or cramps, general convulsions, etc., for details respecting which the reader must consult the other volumes on nervous diseases.

A large group of cases still remain in which no anatomical cause of the pain can be shown to exist, and in which we seem compelled to admit the presence of abnormal conditions of excitation of sensory nerves ramifying on or in the head; these are the cases which may with some propriety be included under the head of "nervous cephalalgia." In these cases the pain may vary considerably in character, and may be described as tensive, tearing, boring, pulsating, and the like. The patient feels as if a red-hot nail were being thrust into his head, as if the head were splitting open, or as if it were being violently squeezed. All shaking of the head, stooping, straining as at stool, unusual exertion, quick movements of the eyes, strong impressions of light or sound, reading, work, augment the intensity of the pain, whilst it is not brought on nor increased by palpation or by tapping upon the head.

The patients often describe very exactly the seat of the pain. It is most commonly situated in the forehead, temporal region, or occiput; but it is frequently stated to be internal and deep-seated, as at the bottom of the orbit, in the internal ear, at the base or in the bones of the skull. It is sometimes unilateral, sometimes bilateral, sometimes fixed, sometimes wandering; in one case it will be diffused over the whole skull, whilst in another it will be limited to one small spot (Clavus).

A great variety of symptoms accompany this form of headache: a feeling of light-headedness and emptiness, of beating within the head, muscæ before the eyes, singing in the ears, fainting, occasionally delirium or slight unconsciousness, convulsions and cramps of certain muscles, slowness of pulse, general malaise and vomiting, and not unfrequently anæsthesia, formication, etc. In addition, symptoms of the primary disease are also discoverable.

The duration of the pain is also extremely variable. It may last for hours, days, and even years, or at least may recur again and again during that period with longer or shorter intermissions (habitual headache). It may remain of moderate intensity, or rise to such intensity as to almost drive the patient to desperation.

Physiology affords little aid in determining the sensory nerves in which the irritation is seated. Experiments upon the sensibility of different structures in the interior of the skull have given contradictory results; for whilst Leyden, Hitzig, and Ferrier have found the dura mater to be highly sensitive, Pagenstecher ascribes a very low degree of sensibility to it, and Bartholow,[1] who experimented on the human subject, found it to be but slightly sensitive to mechanical stimuli. The greater number of experiments seem to show that the pia mater is also not very sensitive, and the same may be said in regard to the greater part of the substance of the hemispheres. When Bartholow pushed his needles deep into the substance of the cerebral tissue, pain was only experienced in the neck after they had been pushed in to about the depth of the great ganglia at the base of the brain.

We are unable, again, from physiological considerations, to explain why headache should be experienced when the intra-cranial disease is situated in so many different parts of the brain or of its membranes; we must either admit that the tissues, which under ordinary circumstances are insensible or are only endowed with a low degree of sensibility, become sensitive under pathological conditions, and give rise to pain; or we may imagine that in diseased states the influence of pressure and irritation spreads to considerable distances, involving tissues that are sensitive. The dura mater may undoubtedly be included amongst such sensitive structures, since it receives sensory branches from the trigeminus and vagus; perhaps also the nerves of the choroid plexus, which Benedict[2] has recently described, are of a sensory nature, and may be answerable for the "internal" headache sometimes complained of. To what special locality, however, we should refer these "nervous" headaches, is still undecided, and we can only offer guesses at the truth.

Various forms of nervous headache may be distinguished in accordance with their *pathogenesis;* these will here be briefly mentioned.

Anæmic Headache.—This usually affects equally the temples and brow, and frequently also the vertex, extending along the sagittal suture, and is dull and tensive in character, and not very violent. It is alleviated by the recumbent position, rest in bed, etc., and is rendered worse by long maintenance of the erect position. It is accompanied by dizziness, a disposition to faint, general pallor, palpitation of the heart, dyspnœa, cardialgic and dysmenorrhœic disturbances, and is frequently met with in chlorotic patients, and in all anæmic patients, especially in women.

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[1] *Bartholow*, in Beard's Archives of Electrology and Neurology. New York, 1874, p. 68.

[2] *Benedict*, Virchow's Archiv, Band 59.

Headache from Active Hyperæmia (congestion) usually affects the whole head, is often accompanied by throbbing, and by sensations of pressure and weight in the head, agitation, hyperæsthesia, and illusions of the organs of special sense; the eyes and face are suffused, and there is strong pulsation in the carotids. Eulenburg has described, under the term *cephalæa vaso-motoria*, violent pain in the brow and temple, which is accompanied by a sense of pressure and tightness in the head, and by heat and redness of the face and ears, and which may come on in regular paroxysms. I have myself seen a case of this kind in which violent pain at the crown of the head, and fainting, were coincident with intense redness and heat of the brow and vertex.

Under the name of *toxic headaches* we may include those caused by the entrance of certain poisons into the system, or by infection with certain products and germs of disease. The best example of this is found in the headache following alcoholic intoxication, which causes, in the morning after free libations, a feeling of painful pressure and weight, which appears to be chiefly localized at the base of the brain and in the deeper parts of the eyes. From the well-known relations of chronic alcoholism to the diseases of the dura mater, there is some reason in referring this form of headache to the sensory nerve fibres of the dura mater. Chronic lead poisoning also often causes cephalæa; poisoning with narcotics, with carbon vapor, sulphuretted hydrogen, etc., frequently leaves behind a long-enduring headache. In many persons, exposure to bad air, and to the air of overcrowded rooms produces headache; violent headache, again, is a common symptom in uræmic intoxication. It is, perhaps, justifiable to apply the term infectious headache to that form

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which accompanies infection of the system with the poison of typhoid fever, malaria, etc. Fournier[1] describes a very severe form of headache, which occurs with special frequency in syphilitic women; it belongs to the secondary symptoms, and differs essentially from the headache caused by syphilitic affections of the bone, and syphilitic neuralgia. These several forms of headache may be distinguished by the concomitant symptoms of the particular kind of poisoning or infection to which they are due.

Headache is one of the most common complaints of *hysterical patients*, in whom it very frequently assumes the form of clavus, the pain being limited to one small spot of the skull; but frequently, also, it is more diffused and deeply seated, extending horizontally from the forehead to the neck, and accompanied always by other symptoms of this Protean disease. The pain is augmented during the menstrual period, by harass and worry of mind, whilst it is removed by amusement and mental excitement.

By *rheumatic headache* is meant the tearing, and often very violent, pain localized in the muscles of the head and in the fascia of the occipito-frontalis muscle; it is generally believed to be brought on by exposure to cold, is often associated with well-marked tenderness of the scalp, and is increased by changes in the temperature and weather. Mettauer[2] has reported a remarkable case of this kind under the title of "neuralgia of the occipito-frontalis muscle," in which he effected a cure by making a subcutaneous crucial incision over the whole cranium.

A very obscure form of headache is that denominated *sympathetic headache*, which may supervene upon diseases of all possible peripheral organs, and is not readily explained. It is most commonly attendant upon diseases of the digestive organs (browache in gastric catarrh, headache from the presence of worms in the intestinal canal, from hemorrhoids, etc.) and of the sexual organs (headache from Onanism, sexual excesses, and from diseases of the uterus and ovaries).

Lastly, there is a form of headache, which I shall take the liberty of calling *neurasthenic headache*, in which the pain is of a heavy, dull, oppressive, and deep-seated character, seriously impairing mental activity and often increasing to the height of migraine. It occurs in those who are depressed by severe mental or bodily exertion, night watching, anxiety, great excitement and sorrow, sexual excesses, etc., and whose nervous systems present manifold indications of diminished resistance to excitation. This is a tolerably frequent and very harassing form of the disease.

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[1] *Fournier*, Leçons sur la Syphilis. Paris, 1873, p. 761.

[2] *Mettauer*, Boston Med. Journal, July, 1870.

In addition, we not unfrequently meet with very puzzling cases, in which literally no cause can be discovered for the extremely violent and tormenting pain so often experienced. In one case which fell under my care, in which the patient was a young waiter, I observed that there was a spot about the size of a shilling on the right side of the skull, in which pain recurred with frightful violence every evening. It was increased by pressure, by stooping, and by movement; but I was unable to discover any cause for it. The fascia of the occipito-frontalis was tender to the touch, but not swollen or red; the nerves distributed to it were not painful, and he had not suffered from any syphilitic affection. The pain in this case was relieved by the stabile application of the anode of the galvanic current on three occasions. In two other cases I observed violent and persistent frontal headache, with reflex wrinkling of the brow, without being able to discover any cause of the affection, or to afford any relief by means of the various remedies which I prescribed. Every busy practitioner must frequently have such cases fall under his observation.

The *diagnosis* of the several forms of headache may be deduced from the foregoing account, and from a consideration of all the concomitant circumstances of the case. The examination should be carefully directed to the external tissues of the head, and to the behavior of the peripheral nerves, the sensory organs, and the functions of the brain. The skull should be percussed, the breath held, and an effort at straining made; the state of the circulatory and digestive organs should be investigated, and the existence or absence of any hereditary predisposition or toxic influences ascertained. Particular attention should be paid to the more obstinate forms of headache, which, when lasting for years together, are often the precursors of serious cerebral disease.

The *treatment* requires much intelligence and tact on the part of the physician, and often calls forth all his resources. In the first place it should be directed to the primary or constitutional disease, and hence the remedies in common use against anæmia, hysteria, syphilis, nervous debility, local diseases, etc., must be employed.

In many cases, however, remedial measures must also be prescribed for the headache itself. The number of remedies that have been recommended for this purpose is of course very great; of these we shall here only mention the following: *cold* in the form of wet compresses, ice bottles, etc., which is most appropriate in the treatment of the congestive, and the majority of the toxic forms, but may also prove serviceable in hysterical and allied states. It may be applied also in the form of cold to the neck, cold foot-baths, cold bandages to the calves of the legs, and cold hip-baths. *Narcotics* have been tried in all their forms, but they do not prove very effective, and it is not easy to lay down any definite indications for their use.

The reader may refer to what has already been stated in speaking of the neuralgiæ. A dose of morphia in a cup of strong coffee often proves very serviceable, and relief may be obtained by placing a little pad of cotton-wool moistened with chloroform upon, or by rubbing veratria ointment into, the affected spot.

Derivatives in various forms may be tried, such as flying or suppurating blisters on the neck or behind the ears, sinapisms, embrocations with ointments that will produce a crop of pustules over the painful spot, moxæ, and even the actual cautery; in some instances leeches may be applied to the temple, or neck, or to the anus.

Electricity is a very valuable remedy in these cases, and may be used in full accordance with the rules already laid down in speaking of the neuralgiæ. The transmission of the galvanic current through the head from before backwards, and from side to side, and along the sympathetic nerve, is of the highest value, and after this the stabile application of the anode to the skull or other painful part. I am able, from personal observation, fully to corroborate the statements of Benedict and others, as to the extremely favorable effects obtained from the application of the "electric hand," that is to say, from faradizing the head by means of the moistened hand of the physician, which constitutes one electrode. The current should be weak, and may be applied for a period of from two to five minutes. The "general faradization" of Messrs. Beard and Rockwell also frequently appears to act well, especially in hysteria, neurasthenia, and allied conditions.

When spasm of the vessels is believed to be present, *nitrite of amyl* may be tried. If, on the contrary, it is believed that there is paralysis, the *ergot of rye* may be prescribed. Eulenburg has obtained excellent results from ergot prescribed in doses of from nine to fourteen grains per diem in his cases of cephalæa vaso-motoria.

Iron, quinine, arsenic, iodide and bromide of potassium may all occasionally be employed, and I strongly recommend a combination of quinine, in doses of one and a half grains, with three-fourths of a grain of powdered digitalis, taken every morning for a considerable period. I only mention nitrate of silver, with or without sal ammoniac, because it has been highly approved of. Trousseau recommends the application of hot sand-bags, at a temperature of from 104° to 122° F., to the head, for half or three-quarters of an hour, though he has not laid down any definite indications for their employment. Chapman states that they may also be applied with advantage to the neck.

In many cases more good can be done by ordering change of air and scene, travelling to baths or for pleasure, and careful regulation of the diet, mode of life, and occupation, than by prescribing all the remedies in the pharmacopœia.

c. *Neuralgia in the Region of Distribution of the Four Lower Cervical Nerves (Plexus Brachialis).—Cervico-Brachial Neuralgia.*

Valleix, loc. cit., pp. 225–272.—*Bretschneider*, *Romberg*, *Hasse*, *Eulenburg*, loc. cit.—*Fil. Lussana*, Monograf. delle Nevralgie brachiali, Gazetta med. ital. Lombard., 1858.—*Bergson*, Nevralgia brachiale, Annal. Universal.. 1860.—*Schramm*,

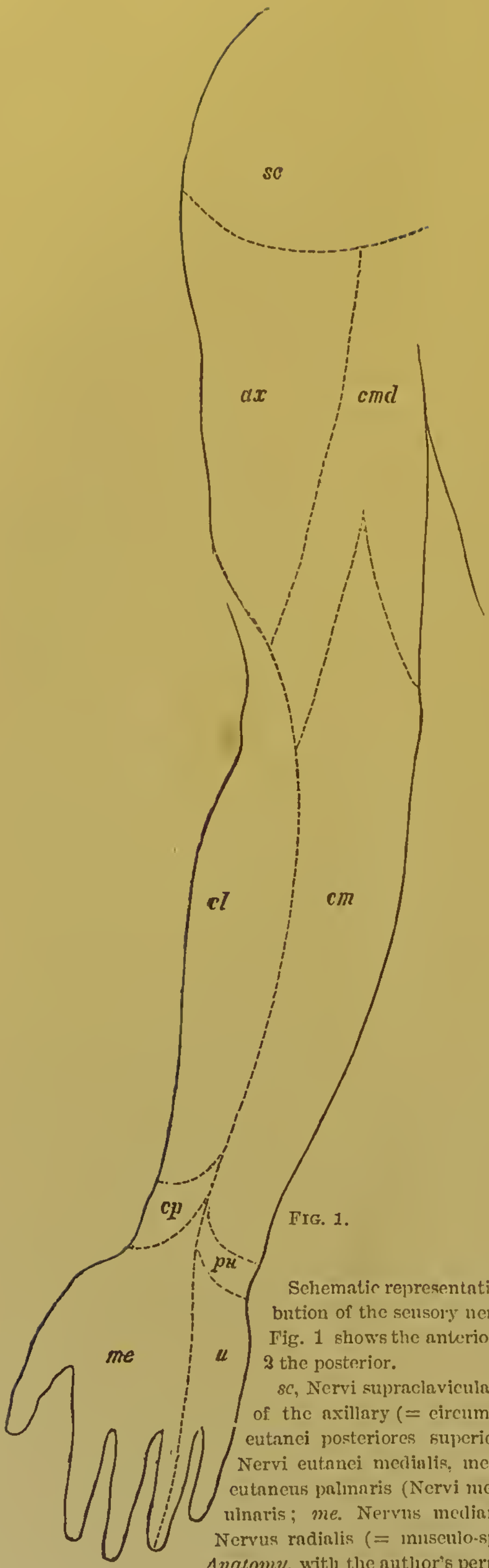

FIG. 1.

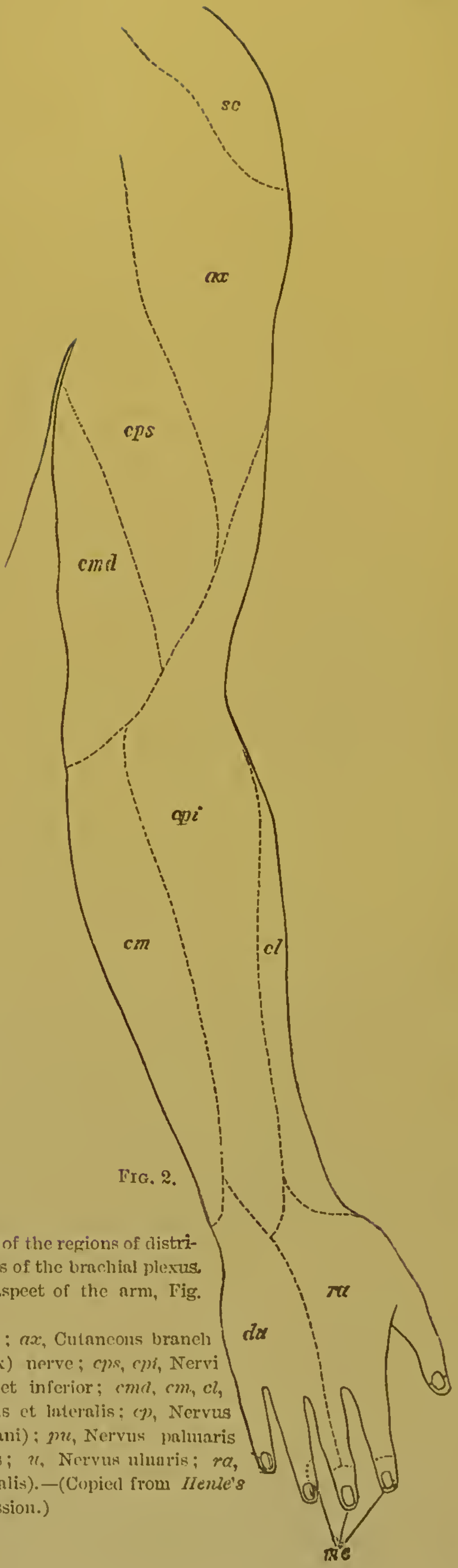

FIG. 2.

Schematic representation of the regions of distribution of the sensory nerves of the brachial plexus. Fig. 1 shows the anterior aspect of the arm, Fig. 2 the posterior.

sc, Nervi supraclaviculares; *ax*, Cutaneous branch of the axillary (= circumflex) nerve; *cps*, *cpi*, Nervi cutanei posteriores superior et inferior; *cmd*, *cm*, *cl*, Nervi cutanei medialis, medius et lateralis; *cp*, Nervus cutaneus palmaris (Nervi mediani); *pu*, Nervus palmaris ulnaris; *me*. Nervus medianus; *u*, Nervus ulnaris; *ra*, Nervus radialis (= musculo-spiralis).—(Copied from *Henle's Anatomy*, with the author's permission.)

Die Neuralgie des Armgeflechts, Bayr. ärztl. Intelligenz-Blatt, 1859, No. 47.—*M. Rosenthal*, Neuralgie des Plexus cervico-brachialis, Wien. allgemein. med. Zeit., 1864, Nos. 7 and 8.

General Characters of the Disease.—Under the term "cervico-brachial neuralgia," all those neuralgiæ are included which have their seat in the sensory fibres of the four lower cervical nerves and a part of the first dorsal nerve, and which may, therefore, affect either the posterior branches of these nerves (which are distributed to the back of the neck), or the several sensory branches of the brachial plexus. In the latter case the pain may be seated in the upper arm (region of distribution of the nervus cutaneus humeri of the axillary nerve,[1] of the cutaneus posterior superior and cutaneus posterior inferior of the radial nerve, and of the cutaneus medialis, which last chiefly supplies the inner surface of the upper arm), or it may affect the forearm (the inner surface of which is supplied by the cutaneus medius and cutaneus lateralis, but partly also by small branches of the median and ulnar, whilst the skin of the back of the arm is principally supplied by branches of the cutaneus posterior inferior of the radial nerve, and by those of the cutaneus medius); or lastly, it may extend into the hands and fingers, affecting the cutaneous branches of the median (supplying palm of hand and palmar surfaces of the three first fingers, and half the ring-finger, and the dorsal surfaces of the last phalanges of the same fingers), of the ulnar (supplying the palmar surface of the remaining half of the fourth and of the fifth finger, and median half of the dorsal surface of the hand and fingers), and of the radial nerve (supplying the lateral half of the dorsal surface of the hand and fingers, and ball of the thumb).

It is not in general possible to draw any sharp lines of division amongst the brachial neuralgiæ, in accordance with the distribution of these nerves, because not only are several of them almost always coincidently affected, but the interweaving of the fibres of the several nerves in the brachial plexus is so intimate and manifold that each of the cervical nerves invariably supplies fibres to several of the peripheric nerves. It is, on this account, not very easy to determine, from an examination of the neuralgically affected parts in the periphery, what fasciculi of the plexus, or even what special roots of the nerves, are implicated. It is only when the primary disease affects some part very near the periphery that the neuralgia is limited to any particular branch of the brachial plexus; if the disease have a higher seat in the plexus itself, or in the several cervical nerves or their roots, we must, as a rule, expect to meet with a wider distribution of the pain affecting a greater number of, and more diverse, cutaneous branches, and this is in accordance with actual facts; the pain is almost always more or less irregularly distributed over many branches, and it can only be said that all possible combinations, though with varying frequency, occur. In this way, the skin lying over the great nerve trunks in the arm and forearm may be painful, whilst the trunks themselves are unaffected, and it is prudent to bear this always in mind, in order to avoid falling into errors in regard to the exact seat of the neuralgia.

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[1] Henle's nomenclature is here followed, as given in his Handbuch der Nervenlehre. Braunschweig, 1871. Henle gives on p. 504 a very convenient systematic representation of the region of distribution of the sensory nerves of the brachial plexus, which is very useful in the study of these forms of neuralgia.

In view of this wide and irregular distribution of the several forms of brachial neuralgia and their various combinations, we can place but little practical importance on the special classification made by Lussana, in accordance with the several nerve branches. On the other hand, it may be of scientific interest to test a great number of exactly observed cases by such a classification, as it may hereafter prove of importance for the more exact localization of the disease.

Etiology.

The predisposing causes are the same as in cases of cervico-occipital neuralgia. Females seem to be more frequently attacked than males, which is in accordance with the fact that hysteria and anæmia appear to be especially strong predisposing causes.

There are numerous *accidental causes* that act with variable intensity, and are better known. Amongst these *injuries* and *foreign bodies* are the most prominent, and have, in many instances, been ascertained to be exciting causes of brachial neuralgia. It is only necessary briefly to enumerate them. They are injuries of all kinds : penetrating and punctured wounds, gunshot wounds, contusions, luxations and subluxations, wounds made in venesection, burns, contusions, foreign bodies of all kinds capable of exciting irritation in the nerves, all forms of tumors, the pressure of aneurisms, the compression exerted by the callus of badly united fractures, affections of the vertebræ, etc. Amongst the diseases affecting the nerves themselves are *neuritis* and *neuroma* (for example, the cicatricial neuroma occurring after amputation).

Exposure to cold, such, for example, as working in cold water, or sleeping on damp ground or on a moist sheet, is a very frequent cause ; *over-exertion of the arm*, especially in the more delicate kinds of work, as pianoforte playing, sewing, and other feminine occupations, have also been regarded as causes. J. Salter has demonstrated *reflex irritation* of carious teeth to be an occasional cause of brachial neuralgia. *Lead poisoning* and *malaria* often produce this form of neuralgia. The relation of *angina pectoris* to brachial neuralgia, which is a common symptom in attacks of angina, cannot be very clearly laid down. Lastly, brachial neuralgia may be a symptom of disease of the cerebral portion of the nervous system, as of hemiplegia, tabes, progressive muscle-atrophy, etc.

Symptoms.

The pain in brachial neuralgia is usually described as being extremely violent, and of a continuous dull, boring, burning character. It is frequently compared to the sensation caused by a severe blow on the ulnar nerve, just above the point where it runs along the internal condyle ; violent, tearing, and lancinating pains are also experienced, which shoot through the arm, sometimes in the ascending, sometimes in the descending direction, often following with great exactness the course of the principal nerve trunks. The frightful burning pain, termed "causalgia" by W. Mitchell, which is frequently complained of in gunshot injuries of nerves, and which was often observed in the late war, is a very remarkable symptom. Similar pains may be experienced in central neuralgiæ; peculiar lancinating pains are, however, more frequent in these affections.

The pain invariably presents distinct exacerbations and remissions, or even complete intermissions, and often occurs in *nocturnal paroxysms*, which last through the night. They are not unfrequently induced or increased by slight movements of the arms, the warmth of the bed, and especially by muscular exertions. The paroxysms may be almost certainly induced, or relapses may be produced, by playing on the piano, by sewing, or other manual exertions.

The *seat of the pain* corresponds to the region of distribution of one or more of the above-mentioned nerves, and requires no further description; what particular nerves are chiefly affected can in general be ascertained without difficulty by careful examination, though it may not be possible to draw the limits very accurately, partly on account of the peculiar central origin of these nerves, and partly on account of their numerous peripheral anastomoses.

In accordance with this, the position of the *painful points* is somewhat indefinite; though very numerous, they are only discoverable on careful examination, and besides, they often shift their position and are more marked at certain times than at others. Such points are most frequently found over the brachial plexus itself, especially from the axilla outwards; at the lower angle of the scapula (which is difficult to explain); on the posterior surface of the shoulder (corresponding with the axillary nerve); on the median at the elbow; on the points of emergence of the cutaneus medius and cutaneus lateralis from the fascia of the forearm; on the ulnar above the internal condyle, and at the wrist; on the radial where it winds round the humerus, and above the wrist; and, lastly, on the spinous processes of the four lower cervical, and two or three upper dorsal vertebræ, and at their sides where the posterior branches appear under the skin. There are, in addition, many other irregular and not exactly defined or definable spots.

Numerous *concomitant symptoms* exist in almost every case of brachial neuralgia. Radiating pains are felt in the region of distribution of the cervical plexus, and of the upper dorsal nerves (hence the frequent complaints of pain in the shoulder and shoulder-blade), and often also in that of the intercostal nerves. A peculiar feeling of stiffness and of creeping in the affected parts is often complained of, whilst in other cases formication and numbness, which frequently correspond to demonstrable anæsthesia, are present. On the other hand, hyperæsthesia often exists in the affected region of the skin, so that the slightest contact, as pressure of the clothes, or of the bedclothes, is unendurable.

The *motility* of the upper extremity is in almost all instances impaired. The movements are rendered stiff, difficult, and painful; the fingers are stiffened, and twitchings and fibrillar contractions are not uncommon ; cramp-like persistent contractions of the various muscles of the arm, however, are less frequently observed. Besides these, we sometimes encounter pareses, and more rarely paralyses of particular muscles and groups of muscles belonging to the affected nerve region.

Coldness and pallor of the arm and hand are amongst the most common *vaso-motor and trophic disturbances*, but in many cases there is abnormal feeling of heat and increased redness. I have occasionally seen a dark-blue redness of the finger, with coldness and free formation of sweat. An eruption of herpes is not unfrequent in the region of the affected nerves. More severe trophic disturbances, as pemphigus, obstinate ulcers, glossiness of the skin of the fingers, changes in the growth of the nails, etc., are usually observed in those forms of neuralgia which are induced by severe injuries to the nerve, and these always accompany serious motor and sensory paralysis.

In regard to the general disturbances, the sections on these in another part of the work may be referred to ; it need only be remarked that, in addition to the brachial neuralgia, symptoms of serious organic disease of the heart and of the large vascular trunks, as well as of the central nervous system, are often found, and these naturally demand special attention.

In its *course*, *duration*, and *terminations*, brachial neuralgia resembles in all essential respects cervico-occipital neuralgia.

The *diagnosis* requires in many cases special care and attention, since, on the one hand, the arm is liable to many kinds of painful affections, which may lead to error, such as muscular and articular rheumatism, diseases of bone, etc., and on the other a sharp limitation of the locality affected by the neuralgia is not very practicable. We must also pay particular attention to the recognition of the characteristic signs and indications of neuralgia generally (see general part), and, on the other hand, we should make every endeavor to ascertain the existence of any local indications of the above-named affections. The diagnosis of the causes of the neuralgia must be made on general principles. The seat of the lesion in regard to its distance from the centre is not easily ascertained, unless, indeed, it affects only a single peripheral nerve, and even under these circumstances it may have a central seat. In regard to the diagnosis of the seat of the disease, in the plexus or in the nerve roots, but few deductions can be drawn from the localization of the pain; precise conclusions can only be arrived at by a general review of all the attendant circumstances.

The prognosis here depends, as in neuralgia generally, upon the cause of the affection, its violence and duration, the constitution and age of the patient, etc.

Treatment.

Treatment directed to the causes of the disease has a tolerably wide field of operation, considering how frequently brachial neuralgia is caused by wounds, foreign bodies, external pressure, etc.; surgical interference is consequently here both practicable and serviceable. In all cases the treatment should be directed on general principles to the removal of the cause of the disease.

I place electricity in the foremost rank in the direct treatment of this affection, the results of its application being in most cases very satisfactory. The galvanic current is usually most serviceable, though one case has fallen under my care in which, after it had been applied without benefit, the faradic current effected a cure. The mode of application must be

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in accordance with general electro-therapeutic principles (see these and the section on the treatment of Sciatica).

In the next place, narcotic remedies cannot be dispensed with. Lussana recommends belladonna or atropine as the best, but we give the preference to morphia, which should be injected subcutaneously in a methodical manner. When this fails, other narcotics may be tried. In mild cases external applications of narcotics are of benefit, such as liniment of chloroform, ointments containing opium and especially veratria, which in my hands, despite the adverse opinion of Eulenburg, has yielded excellent results in many cases. I use it made into an ointment with twenty parts of fat, either by itself, or, what I prefer, combined with extract of opium. Whether the good effects come from its narcotic or its irritant properties, I cannot say.

Counter-irritation applied to the skin is likewise of great value, especially in the form of flying blisters, embrocations with turpentine, mustard oil, etc. Strong vesication applied to the affected part, and repeated till relief is obtained, is particularly recommended by W. Mitchell as a means of alleviating the frightful pain of "causalgia;" and next to this are injections of morphia thrown into the part itself, and followed by persistent cold-water dressing. In the more severe cases transcurrent cauterization should be tried. For *internal treatment* Schramm recommends quinine, and in chronic cases arsenic. Turpentine has also been found useful. Cold or hot water treatment, in the form of baths, etc., should be undertaken, according to the special indications of the case.

Operative measures should only be resorted to in cases where the purely sensitive cutaneous branches are affected. In the case of the mixed nerves, neurectomy should only be performed as a *dernier ressort*, owing to the unavoidable and extremely annoying motor paralysis by which it is followed. In all cases distinct evidence of the existence of a peripheral cause for the neuralgia should be obtained before the operation can be considered justifiable. That this is often possible is undeniable, and an endeavor should be made to secure in due time a union between the divided extremities of the nerve, if we wish to prevent the paralysis from becoming permanent. Very extraordinary circumstances alone, and in fact never the neuralgia *per se*, can justify amputations or resections of the upper extremity.

The appropriate regimen in brachial neuralgia must be laid down on general principles. It need only here be specially noted that rest of the affected limb is of primary importance in securing a recovery; in consequence of which all exertion, especially in the more delicate kinds of work, such as sewing, knitting, writing, etc., should be entirely forbidden.

d. *Neuralgia in the Region of Distribution of the Dorsal Nerves.—Intercostal Neuralgia.*

Chaussier, Table synoptique, etc., 1803.—*Nicod*, Nouveau Journal de Médecine, Chirurgie, etc. Paris, 1818.—*Bassereau*, Essai sur la névralgie des nerfs intercostaux, 1840.—*Valleix*, loc. cit., pp. 272–349.—*Beau*, De la névrite et de la neur. intercostal., Archives générales, 1847.—*Erlenmeyer*, Deutsche Klinik, 1851.—*And. Leoni*, Considérations sur la névralgie dorso-intercostale. Thèse, 1858.—*Bretschneider*, *Romberg*, *Hasse*, *Eulenburg*, loc. cit.

Mastodynia: *A. Cooper*, Illustrations of the Diseases of the Breast. London, 1829.—*Carpentier-Méricourt*, Traité des maladies du sein, 1845.—*Alfter*, Neuralgia mammæ, allgemein. med. Centralzeitung, 1856, No. 25.

General Characters.—Those forms of neuralgia which have their seat in the region of distribution of the sensory fibres of the twelve pairs of dorsal nerves, are included under the term "dorso-intercostal neuralgia." The pain may affect the skin of the back and of the loins, as far down as to the crista ilei, which is supplied by the posterior branches of the dorsal nerves; also the skin of the whole anterior and lateral wall of the thorax and belly, down to the symphysis pubis, to which the cutaneous branches of the intercostal nerves are distributed; and since the first and second intercostal nerves supply cutaneous branches to the axilla and to the inner surface of the upper arm, these parts may also occasionally be the seat of the neuralgia. It is rare to meet with cases in which both the anterior and posterior branches of the dorsal nerves are coincidently affected with neuralgia. Usually it is the anterior ones that suffer, so that in the greater number of cases there is only a "neuralgia intercostalis."

This form of neuralgia is almost always unilateral, and the left side is most commonly affected. The frequency with which the several intercostal nerves are affected differs considerably, the fifth to the ninth being apparently the most liable. As a rule, only one or two nerves are implicated. It is relatively common to meet with combinations of this form of neuralgia with others (superficial as well as visceral), as with brachio- and lumbo-abdominal neuralgia, angina pectoris, and cardialgia.

Etiology.

In regard to *sex*, women are especially liable to it. The tables of Valleix and Bassereau give fifty-one women to eleven men. Those between twenty and forty years of age are most frequently affected; weak, nervous, hysterical, and anæmic subjects are strongly predisposed to attacks of it, as are also those who are convalescent from other diseases.

Amongst the *direct causes*, *catching cold* plays some, though not a very important, part; its action on those who are predisposed to the disease is, however, often very well marked. Blows upon the chest and back, injuries, and wounds occasionally constitute traumatic causes of the disease. *Anatomical changes in the nerves themselves* have seldom been noticed; now and then neuroma, and in some cases neuritis, which is then often associated with herpes zoster, have been observed, and in other cases thickening and induration of the neurilemma. Disease of some adjoining organ is still more important as a cause of intercostal neuralgia. This is especially the case with pulmonary phthisis, which, both in its earliest as well as in the later stages, is not unfrequently accompanied by true intercostal neuralgia (which, however, must not be confounded with the pain of pleurisy). The causal relation between the two affections is not very clear; it may possibly only be due to some collateral hyperæmia of the neurilemma, perhaps to inflammation extending from the pleura, and perhaps to mechanical stretching and compression of the nerves through pleuritic false membranes. *Dilatation of the venous plexus* in the interior of the vertebral canal has also been signalized as a cause; and obstruction of the venous circulation on the left side—which, on this side, from the fourth to the eighth intercostal space, is rendered relatively difficult—has been regarded as a reason for the greater predisposition of the left side (Henle). *Aortic aneurisms*, which lead to absorption of the vertebræ and ribs pressed upon, and either thus, or in some other way, stretch the intercostal nerves in their vicinity, may occasion severe neuralgia. This is true of all kinds of *disease of the vertebræ*, as spondylitis, caries, tuberculosis, cancer of the vertebræ, etc., as well as of *diseases of the ribs* (periostitis, caries, necrosis).

Diseases of the spinal cord, especially circumscribed myelitis, meningitis spinalis, tumors in the vertebral canal and spinal cord, and often tabes dorsalis, very frequently constitute causes of intercostal neuralgia. The pains, which in these diseases encircle the trunk at various heights, like bands or girdles, are due in great part to intercostal neuralgia, and present its characteristic symptoms.

Malarial infection has been occasionally noted as a cause. Uterine diseases, however, have not the great etiological importance which has been ascribed to them by many (Bassereau). The careful inquiries of Valleix have also shown that disorders of menstruation only possess secondary and accidental importance.

Symptoms.

The pain, as already stated, appears to be most frequently seated in the anterior and lateral walls of the trunk (neuralgia intercostalis); more rarely it is located in the back. The affected area corresponds exactly to the number and distribution of the nerves implicated. As a rule, the pain is more or less dull, tensive, and persistent, but interrupted from time to time by tearing, lancinating, burning pains, which may increase to a regular paroxysm. The precision with which darting pains are described as following exactly the course of the nerves from the back to the front of the belly is often very remarkable, and it is even sometimes stated that the pain radiates forwards and backwards from the point of emergence of the lateral perforating branch. Not unfrequently also the sensation of a knife running through the chest is complained of. The violence of the pain may be very great, whether it occur in the form of a ring round the chest, or of lancinating pain. All respiratory movements, such as coughing, sneezing, and blowing the nose increase the pain, as do also slight pressure on the skin, the weight of the clothes, etc., whilst firm pressure sometimes alleviates it. The paroxysms are induced and increased by the ordinary causes. Valleix believes that he has proved that snowy weather is especially harmful. During the paroxysms the patients present a very characteristic appearance; they sit with their bodies inclined to the affected side, and with an anxious expression of face, daring neither to breathe deeply nor to speak loudly.

Characteristic *painful points* are almost always discoverable in intercostal neuralgia. First, there is one close to the vertebral column, corresponding to the point where the nerve emerges from the intervertebral foramen (vertebral point); a second, where the ramus perforans lateralis emerges beneath the skin, that is about the middle of the entire course of the nerve (lateral point); a third, where the perforans anterior pierces the muscles, in other words, close to the sternum; and in the abdomen over the rectus muscle (sternal or anterior point). These points are usually very distinctly demonstrable, and are extremely sensitive to pressure. The whole length of the intercostal nerve, and several of the spines of the dorsal vertebræ, are frequently extremely sensitive and tender.

Radiation of the pain towards the back and arm, and not unfrequently also into the loins and lower extremities, and complications of the attack with angina pectoris, cardialgia, and with colic from gall-stones have been observed. *Hyperæsthesia* of the affected region of the skin is very common, so that even the weight and friction of the clothes cannot be endured. *Anæsthesia*, on the other hand, is less frequent, but has, nevertheless, been distinctly observed, usually affecting a small circumscribed area. This appears to be particularly the case after an attack of zoster. Of *motor phenomena* the only one observed is the interference with the respiratory movements, which is very common, without much being known in regard to its mechanism.

But little, again, has been noted in regard to *vaso-motor and* *trophic disturbances*, with the exception of the occurrence of herpes zoster, which has been very frequently observed. The relation of this affection to neuralgia is, however, very inconstant. Many cases, in fact the majority of cases, of intercostal neuralgia occur without zoster; moreover, cases of zoster not unfrequently occur without neuralgic symptoms, and this, indeed, is the rule in children and young persons; and, lastly, there are cases in which the neuralgia appears associated with herpes, but in which the neuralgia both appears before it, and outlasts it, as is usually the case with old people. It is impossible to deny that a causal relation exists between the two phenomena, but the nature of this cannot be considered as ascertained. The various views on this point have been mentioned in the general part, and we must, therefore, refer the reader to that section. From the foregoing facts, however, one conclusion may very properly be drawn, namely, that where intercostal neuralgia is accompanied by herpes zoster, a neuritis of the nerves may be regarded as the cause of both phenomena. No observations have been made in regard to any alteration of the secretion of the milk in the ordinary form of intercostal neuralgia.

The development of the disease takes place, as a rule, so gradually that the patient is unable to fix the exact date. Its course is extremely irregular, and it is not unfrequently very obstinate. If recovery take place, it is also very gradual. The terminations of the affection are for the most part dependent on the nature of the primary affection. Recovery takes place in the majority of rheumatic and idiopathic cases; in central lesions of the nervous system it occurs only slowly and the patient is liable to relapses; in pulmonary phthisis and disease of the vertebræ, and the like, the disease not unfrequently terminates only with death.

The *diagnosis* of intercostal neuralgia can often only be made with great difficulty. It is most frequently confounded with rheumatism of the thoracic muscles (commonly called pleurodynia). These two diseases are in fact so liable to be mistaken for one another, that they can only be distinguished by the most careful examination, and the diagnosis can in some cases only be ascertained by the mode in which the disease terminates. The seat of the pain in certain muscles, its aggravation on making certain movements, especially of the head and limbs, the absence of painful points in the characteristic situations, and lastly, the rapid recovery in the course of a few days, may serve to distinguish pleurodynia from intercostal neuralgia. Diseases of the thoracic organs are easily distinguished from this form of neuralgia by careful physical exploration. In angina pectoris, which resembles intercostal neuralgia in many respects, the immense anxiety and the feeling of threatened suffocation and impending death, together with the phenomena presented by the heart and pulse, are sufficiently characteristic. The diagnosis of the causes of the disease is a matter of primary importance, especially to the patient, and the greatest care should be taken to determine, at as early a period as possible, whether it is due to any disease of the vertebræ or spinal cord.

The *prognosis* must be in accordance with what has been stated in regard to the causes and course of the disease. Simple rheumatic and idiopathic cases pursue a comparatively mild course as compared with the other forms.

Treatment.

In regard to fundamental principles, the remedies to be used, and the mode of applying them, little can be added to what has already been stated in the general part and in treating of the foregoing forms of neuralgia. After such measures have been adopted as are required to meet the causal indications, the use of counter-irritants to the skin are to be specially mentioned, and amongst the best of these are the flying blisters so strongly recommended by Valleix. These should be applied in succession over the painful points, and their action is often very striking. In slighter cases, milder cutaneous irritants or narcotic and anæsthetic embrocations will be found sufficient. With these, subcutaneous injections of morphia may be advantageously combined, especially in severe cases. Electricity often proves of essential service, partly applied as a cutaneous irritant (faradic brush or moxa), partly acting upon the affected nerves; the best mode of effecting the latter is by the application of the galvanic current in the manner already described (the anode being placed on the vertebral column, the cathode upon the lateral and anterior painful points, and the current being strong and stabile). In regard to the use of specifics, and other methods of cure, the general rules must be followed. Operative measures can only be resorted to in extreme cases.

Simple measures only are required for the treatment of the zoster, which so often accompanies this form of neuralgia. In addition to the treatment of the neuralgia, the surface should be covered with some indifferent and protective ointment or plaster, to which some narcotic may be added. Cauterization of the vesicles is quite superfluous, and the application of vesicants is not advisable. At most, they may be applied to the side of the vertebral column, providing no vesicles have made their appearance at this point; because, according to Anstie's views, vesication over the posterior roots exerts a favorable action on neuralgiæ of the anterior roots. Anstie believes that he has in this way prevented the further development of the herpes.

APPENDIX.

Neuralgia of the Mammary Gland.—Mastodynia.

The nervous supply of the mammary gland is chiefly derived from the intercostal nerves, the skin over it being supplied by the lateral and anterior perforating branches of the second, third, fourth, fifth, and sixth intercostals, and to a small extent also by branches of the supraclavicular nerves, whilst the proper substance of the gland is supplied by the lateral perforating branches of the fourth, fifth and sixth intercostals.

The mammary gland is itself liable to the most exquisite neuralgic pains and paroxysms of pain, and this has led to the formation of a subordinate class of intercostal neuralgia, which was first minutely described by Sir Astley Cooper under the name of "irritable breast." The pain in this affection presents all the characters of neuralgia; it may become frightfully intense, and even approximate the violence of the attacks of tic douloureux. The pain is tearing, cutting, boring, and lancinating, and as though a knife were run through the part; it appears in paroxysms, which are usually of short duration, but may last for several hours. The breast appears much heavier than natural; the patient cannot lie on the affected side; the pressure of the clothes or the slightest contact is absolutely unbearable. *Painful points*, though not well defined in character, may be found on the nipple or on the sides of the breast. On the other hand, the spinous processes of the second, third, fourth, fifth and sixth dorsal vertebræ are usually tender on pressure. The paroxysms are not unfrequently accompanied by vomiting, and there is almost always a high degree of a cutaneous hyperæsthesia. During the height of the attack the pain radiates into adjoining nerve regions, as into the wall of the chest and into the arm, neck, and back. Exacerbations of all the symptoms usually occur during the catamenial periods. No accurate observations have been made in regard to the secretion of milk. I observed in one instance a distinct, though small amount of secretion, which made its appearance after the pain had passed off. Alfter saw one case in which zoster followed the neuralgia. Rufz has recorded a case in which the disease occurred in a man. Hasse found the mammilla swollen and painful in a boy; in other words, inflammation must have set in, which is not very unfrequent in boys. The disease usually persists for years, and often obstinately resists every kind of treatment. Patients with this trouble are subjected to a great deal of suffering.

The etiology of the disease is still rather obscure. In many cases there appears to be some causal relation between it and lactation or pregnancy. The neuralgia frequently follows shrinking of the nipple. Anæmia, chlorosis, and hysteria also play an important part in its production. Amongst local conditions, injuries to the mammary gland are satisfactorily known to be causes of the disease. In certain cases small neuromata, or painful tubercles of the nerves of the mammary gland, appear to be the starting-points of the neuralgia. In such cases small, hard, and very sensitive knobs are perceptible in the gland tissue, which remain unaltered for years, sometimes being very painful and sometimes quite painless, whilst in other instances, and without any obvious cause, they entirely disappear. As far as present observations have gone, these swellings appear to be small fibromata or true neuromata.

Decided difficulties lie in the way of diagnosticating these tumors from the early stages of malignant growths which are often also accompanied by severe lancinating pains; but correct conclusions may be arrived at, after a certain lapse of time, by observing their more rapid rate of growth, their less marked sensitiveness to pressure, and the difference in degree of the pain. Careful examination is, however, required to prevent confounding them with mastitis, circumscribed abscess, etc.

The *treatment* is, in many instances, not very satisfactory. The causal indications must first be attended to, especially concomitant anæmia, as well as disturbances and diseases of the generative organs.

In the way of direct treatment the ordinary anti-neuralgic remedies should be prescribed—narcotics, electricity (from which I have in one case seen excellent results), and specifics. Cooper recommended a narcotic plaster containing belladonna. The abstraction of blood has in many cases proved useful. Enveloping the breast in warm coverings, especially in the form of fur, is generally comforting to the patient. Operative measures, such as extirpation of the painful knobs, and amputation of the breast, can only be undertaken when other measures fail.

e. Neuralgia in the Region of Distribution of the Lumbar Nerves (Plexus lumbalis).—Neuralgia lumbo-abdominalis, cruralis, and obturatoria.

Valleix, loc. cit., pp. 349–388.—*Bretschneider*, *Romberg*, *Eulenburg*, loc. cit.—*Neucourt*, De la Névralgie lombaire, Archives générales, 1858.—*Bousseau*, Deux observations de névrose du nerf saphène externe, Gaz. des hôpitaux, 1869. No. 7.—*Liegey*, Intercostal und Hodenneuralgie mit neuralgischem Priapismus, Presse med., 1855, No. 37.—*D'Axthrey*, Considérations sur quelques formes de la névralgie lombo-abdomin.—Thèse de Strassburg. 1867.—*Röser* und *Rotteck*, Uber Hernia obturator, Archiv f. phys., Heilk., 1851.—The literature given under the head of Sciatica, contains many references to works bearing on this subject.

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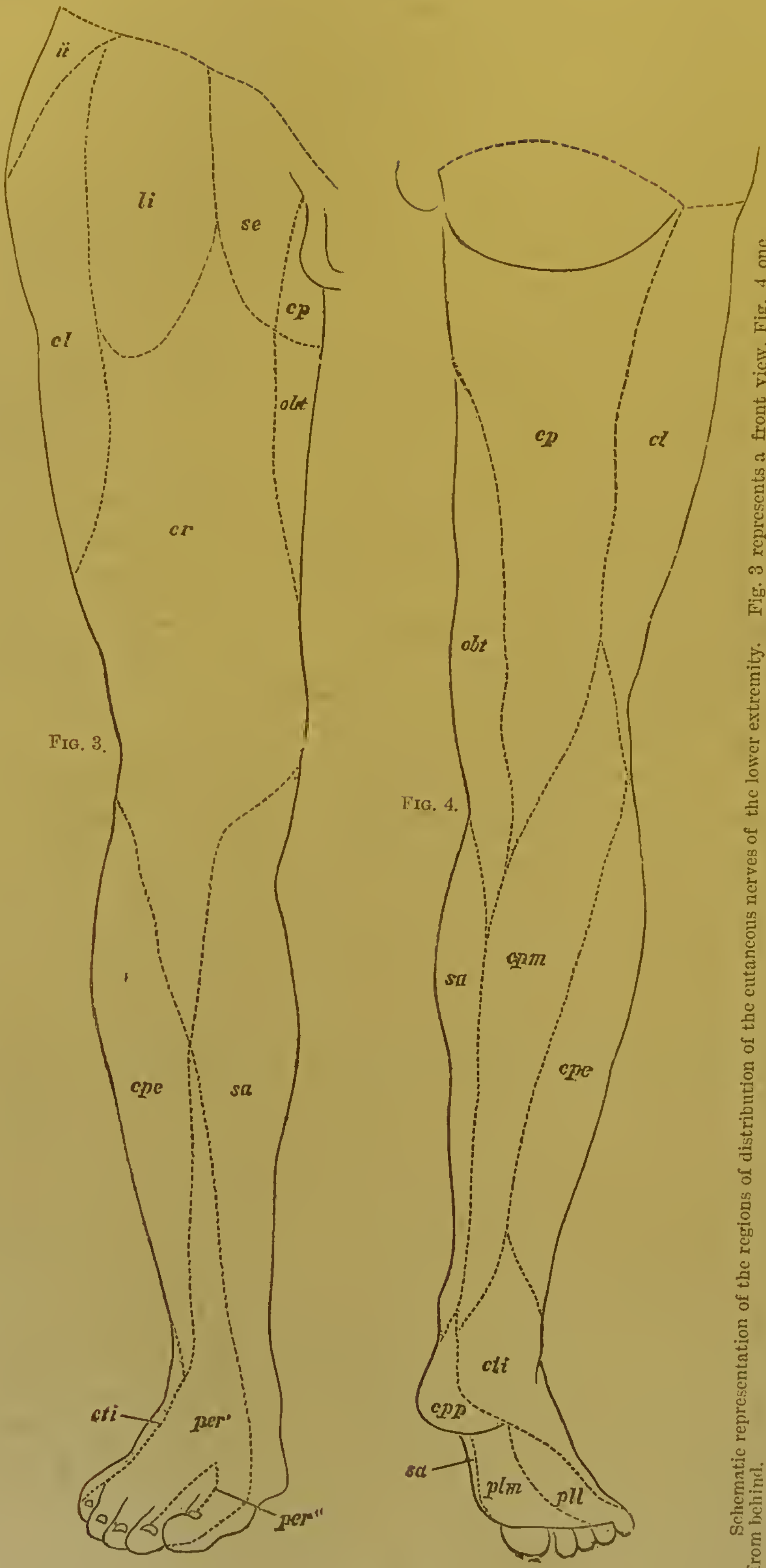

Fig. 3.

Fig. 4.

Schematic representation of the regions of distribution of the cutaneous nerves of the lower extremity. Fig. 3 represents a front view, Fig. 4 one from behind.

ii, Nervus ilio-inguinalis; *li*, Nervus lumbo-inguinalis; *se*, Nervus spermaticus externus; *cp*, Nervus cutaneus posterior; *cl*, Nervus cutaneus lateralis; *cr*, Nervus cruralis; *obt*, Nervus obturatorius; *sa*, Nervus saphenus; *cpe*, Nervus communicans peroneus; *cti*, Nervus communicans tibialis; *per'*, *per''*, Nervi peronei rami superficialis et profundus; *cpm*, Nervus cutaneus posterior medialis; *cpp*, Nervus cutaneus plantaris proprius; *plm*, *pll*, Nervi plantares medialis et lateralis. —(Copied from *Henle's Anatomy*, with the author's permission.)

General Characteristics of the Disease.—All neuralgiæ having their seat in the sensory fibres of the first four pairs of lumbar nerves are included in the above heading, as it is impossible to make any distinction on anatomical grounds between them. The anastomoses and the varieties of origin of these nerves are so numerous, and their mode of distribution so different in different individuals, that an exact analysis of the several forms would lead to minutiæ that would be in no way justified by their practical importance.

The following are the principal cutaneous regions which, as a rule, are supplied by the lumbar nerves: the region of the loins (posterior branches of the lumbar nerves), the gluteal region, inguinal region, hypogastrium and mons veneris, part of the scrotum (or labia majora), the anterior lateral and median surface of the thigh, the anterior region of the knee-joint, the median surface of the leg, and the inner border of the foot as far as to the great toe. This extensive region may be affected to a greater or less extent in different cases, according to the height at which the nerves are affected. Such differences are, however, of no practical importance.

It scarcely ever happens that the whole of the sensory fibres of the lumbar plexus are coincidently affected with neuralgia; for the most part only single or a few branches are implicated, and we may, for the sake of convenience, divide the different forms into two principal groups, of which the first includes neuralgiæ of the so-called (by Henle) "short" nerves of the lumbar plexus, which we shall designate by the general term *lumbo-abdominal neuralgia.* The second group includes the neuralgiæ of the so-called "long" nerves of the crural plexus, which may be indicated by the general term *crural neuralgia.* The posterior branches of the lumbar nerves may contribute fibres to both groups of nerves, though usually to one of them only to a very slight extent.

Lumbo-abdominal neuralgia includes the neuralgiæ of the ileo-hypogastric nerve (affecting the skin of the hip and upper part of the hypogastrium), the ileo-inguinal nerve (affecting the skin over the tensor fasciæ and mons veneris), the lumbo-inguinal nerve (affecting the median portion of the inguinal region as far as to the middle of the thigh), and the external spermatic nerve (affecting the scrotum or labium majus, and adjoining skin of the inner surface of the thigh). It would be superfluous to distinguish each of the neuralgiæ affecting these several nerve regions by a special name.

Three varieties of crural neuralgia are possible, according to the nerves that are chiefly or exclusively affected. The nervus cutaneus femoris lateralis may be the seat of the pain, which then affects the outside and part of the back of the thigh, as far down as to the knee; or the nervus cruralis (with the nervus saphenus) may be affected, the pain then being in the skin of the anterior and internal surface of the thigh and knee, and of the inner side of the leg and foot; or, lastly, the nervus obturatorius may be implicated, the pain then being seated in the inner side of the thigh and extending as low as to the knee. As these nerve regions are tolerably sharply defined from one another, the several neuralgiæ affecting them may very well receive distinct names.

All these forms of neuralgia are comparatively rare. Lumbo-abdominal neuralgia is commonly associated with intercostal neuralgia, which it resembles in affecting the left side, and the female sex principally; whilst crural neuralgia is more frequent in males. Of these several forms, the neuralgia cruralis is the most common.

Etiology.

The etiology of these forms of neuralgia is at present but little known. In fact, we know nothing of the special causes that predispose to it, nor anything beyond what has already been said in regard to the etiology of neuralgiæ in general. Catching cold is one of the commonest causes, next to which comes over-exertion, which, in the working classes, is very frequently followed by crural neuralgia. Injuries (as gun-shot wounds, etc.), foreign bodies (kernels of grain, and the like), neuromata and other tumors have been observed to constitute causes of the disease. Compression of the nerves from hernia (as crural hernia and obturator hernia), or from constipation and accumulation of fæces, is a cause of great practical importance. Carcinoma of the pelvis and of the vertebral column, diseases of the vertebræ, psoas abscess, diseases of the uterus and vagina, have been ascertained to be causes. The violent pain in the knee in coxitis must also be regarded as neuralgic, though the mode of its production is still somewhat obscure. It may, perhaps, be regarded as a phenomenon of radiation (reflex neuralgia), or it may be due to the mechanical pressure of the swollen hip-joint, or to the reflex contraction into which the psoas muscle is thrown. According to Mauriac, orchitis and blennorrhagic epididymitis are not infrequently causes of lumbo-abdominal neuralgia. Lastly, spinal diseases, such as meningitis and myelitis spinalis, and especially tabes, are very frequently accompanied by excentric neuralgiæ in these nerve regions also.

Symptoms.

In view of the rarity and small practical importance of these neuralgiæ, we must limit our observations to the consideration of the most important points.

a. Lumbo-Abdominal Neuralgia.—The point which is especially characteristic of the forms of neuralgia belonging to this group is the combination of pain in the loins, which may extend over the crista ilii as far as to the buttock, with pain in the hypogastrium, mons veneris, and scrotum, or labia majora. The inguinal region is less frequently affected at the same time; still it does occur, and from what has been stated above in regard to the distribution of the several nerves, it will be easy to explain satisfactorily the seat and extent of the pain.

The pain is of the kind usual in neuralgia—dull, aching, and persistent—but interrupted now and again by paroxysms of tearing, boring, lancinating sensations, which often extend from the loins forwards towards the parts chiefly affected. The violence of the pain is rarely intense; it is usually moderate.

Painful points are very commonly found in this group of neuralgiæ: thus there may be one or several in the lumbar region near the spinal column (lumbar point); one about the middle of the crista ilii (iliac point); one or several above the symphysis pubis, at the side of the linea alba (abdominal point); not unfrequently one in the scrotum or labium majus; and lastly, and less constantly, one in the inguinal region, or in the portio vaginalis uteri, or in the corresponding side of the vault of the vagina.

It is often observed that the pain radiates into the neighboring nerve regions, and especially into those supplied by the crural and intercostal nerves, and it is probable that the sympathetic plexuses of the pelvic organs are often implicated.

Of motor disturbances I find only one mentioned in reports of such cases, viz., a difficulty in walking, consequent upon the pain; in a few cases cramp of the cremaster has been noticed. Few observations are to be found in the literature of the subject in regard to any other concomitant phenomena. Vomiting is not unfrequent during the paroxysms. I have observed zoster in one of my own cases. Notta mentions having met with cases in which there were increased sexual desire with priapism and ejaculation of seminal fluid, increased desire to pass water, and pain in micturition, fluor albus. Further researches are, however, needed to establish the exact relations of these phenomena with the neuralgic affection.

I must not omit to state here, that Valleix, d'Axthrey, and others regard the neuralgia spermatica (irritable testis), described by Sir Astley Cooper, as a subordinate kind of lumbo-abdominal neuralgia (ileo-scrotal neuralgia), whilst Romberg, A. Eulenburg, and others locate it in the plexus spermaticus sympathetici. The numerous anastomoses of the spinal and sympathetic trunks in this region render it impossible to decide this point on anatomical grounds, and at the same time afford an explanation of the manifold combinations and modifications of the phenomena presented by the disease. We shall briefly notice again the neuralgiæ affecting the external genital organs.

b. Femoral Neuralgia.—Under this term are included three different forms:

1. *Neuralgia of the Nervus Cutaneus Femoris Lateralis.*—The pain in this variety of neuralgia extends down along the outer, and, to a certain extent, also over the posterior side of the thigh, as far as to the knee. As the branches of this nerve are distributed chiefly over the posterior parts of the thigh, care should be taken not to mistake this form of neuralgia for that

of the nervus cutaneus femoris posterior, whose branches are distributed to the region which is also supplied by the nervus ischiadicus. The only constant painful point is over the anterior superior spinous process of the ileum, where the nerve emerges from the pelvis; other painful points are sometimes found along the outer side of the thigh. The character of the pain and the concomitant symptoms are the same as in the following form.

2. *Neuralgia of the Nervus Cruralis.*—The region affected in this form of neuralgia is the middle and inner part of the anterior surface of the thigh, the anterior surface of the knee, the inner surface of the leg and of the foot as far as to the great toe. The pain in the lower leg and internal border of the foot is highly characteristic of the disease, pointing as it does to the implication of the saphenous nerve. The pain is of a tearing, boring, lancinating character, shifting from one point to another, and is usually most severe at night; in the intervals there is only a dull feeling of pain. Movements of the leg, whether active or passive, but especially those of locomotion, cause considerable increase of pain.

Painful points are very commonly met with in the fold of the groin, at the point where the nerve emerges from the pelvis; on the inner side of the knee-cap, where the nervus saphenus appears beneath the skin; in front of the ankle bone; and at the base of the great toe. Inconstant points, which are in the majority of instances difficult to find, and only transitory, are found on the thigh, where the smaller cutaneous branches are given off. Radiation of the pain occurs into the other branches of the lumbar plexus, the posterior branches of the lumbar nerve, etc. Hyperæsthesia of the skin is a very common symptom, especially in the vicinity of the knee-joint.

Anæsthesia, partial in extent, but more or less complete in degree, also occurs. A sense of formication is often experienced in the region of distribution of the saphenus, and less frequently a feeling of numbness. The motor disturbances are generally very marked: the patient complains of weariness and pain in walking, weakness and paresis of the muscles of the thigh, etc. Bousseau saw redness, swelling, and increased secretion of sweat along the inner border of the foot, in a case of neuralgia of the saphenus, and I have observed in one case a high degree of atrophy of the anterior muscles of the thigh. Whether the hysterical neuroses of the knee-joint, first described by Brodie, with the concomitant phenomena of redness of the skin, swelling, and augmented heat, are forms of crural neuralgia, must be at present regarded as an open question (see below, Articular Neuralgia).

3. *Neuralgia Obturatoria.*—In this form the pain is confined to the inner side of the thigh, extending as far as to the knee-joint. It is an uncommon affection, and has only this practical importance, that it may furnish some assistance in making the diagnosis of the difficultly recognizable obturator hernia, whether strangulated or not. Romberg was the first to call attention to the fact that in difficult cases of this kind the diagnosis can be made from the presence of symptoms of incarceration, together with neuralgic pains in the region of distribution of the nervus obturatorius. Formication on the inner surface of the thigh, and a feeling of stiffness and immobility of the adductors are usually present. In other respects this form of neuralgia presents no special points of interest.

The course and duration of all these forms of neuralgia vary considerably, in accordance with the cause of the affection; experience teaches that as a rule the disease is of short duration, and pursues a mild course, though this holds only in the case of those forms that are uncomplicated with any severe organic disease. On this, of course, the events, as well as the prognosis, of the affection primarily depend. In simple forms the *prognosis* is tolerably favorable; they usually get well quickly and perfectly.

The diagnosis presents in many cases considerable difficulties. Lumbago is apt to be mistaken for the first group, but error may be avoided if attention be paid to the circumstances that in lumbago the pain is usually localized in one sharply circumscribed point; that it does not shoot forwards; that bending forwards, stretching the body, raising a weight, are all particularly painful, and that pain disappears when absolute rest is maintained. The pain in cases of renal calculi cannot in many cases be distinguished from that of neuralgia, and is due in part to radiation to the spinal nerves. The diagnosis of the second group from hip and knee-joint diseases is often very difficult, and a right conclusion can only be arrived at by careful examination. A circumstance that is worthy of special attention in such cases is, that in neuralgia the skin is more sensitive than the joint itself.

It is unnecessary to enter into any special details in regard to the *treatment*. The usual measures must be taken to remove the cause; flying blisters are useful in the slighter cases; injections of morphia and electricity for the more obstinate ones. In three cases of crural neuralgia I have quickly attained excellent results from the use of the galvanic current (descending stabile current made to pass from the lumbar region of the vertebral column to the nerve and likewise through it). Bousseau was successful with injections of atropine when morphia had failed. It is seldom requisite to resort to more energetic measures, but for the treatment of obstinate and severe cases the reader is referred to the next section on the treatment of sciatica.

f. *Neuralgia in the Region of the Sacral Nerves* (*Plexus Sacralis*).—*Neuralgia ischiadica.—Sciatica.—Malum Cotunnii.* —(*Hüftweh*).—*Neuralgia pudendo-hæmorrhoidalis.*

Dom. Cotugno, Comment. de ischiade nervosa. Neapol., 1764.—*Petrini*, Nuovo methodo di curare la sciat. nervos. Roma, 1781.—*Lentin*, Hufeland's Journal I., 1795.—*Martinet*, L'emploi de l'huile de térébinth dans la sciatique, 1824.—*Fioravante*, Annal. universal, 1843.—*Baruch*, Natur und Behandlung d. Ischias. Oesterrich. med. Jahrb., 1845.—*Valleix*, loc. cit., pp. 388–520.—*Robert*, Traité de la Sciatique, etc., Rev. méd., 1847.—*Jobert*, Union méd., 1859, No. 77.—*M. Rosenthal*, Wien. allg. med. Zeitung, 1864, Nos. 11–14.—*Fuller*, Clinical Lectures on Sciatica, Lancet, 1864.—*Betz*, Zur Pathologie und Therapie d. Ischias. Memorab., 1865.—*Lagrelette*, Étude histor. semiolog. et thérapeutique de la Sciatique. Thèse, Paris, 1869.—*Patruban*, Blosslegung und Dehnung des grossen Hüftnerven behufs Heilung der Ischialgie, Allg. Wien. med. Zeit., 1872, Nos. 43, 44, 47, 53.—See also *Romberg*, *Hasse*, *Eulenburg*, and the text-books on Electro-therapeutics.

D'Axthrey, Considérations sur quelques formes de la névralgie lombo-abdom. Thèse, 1867.—*Liegey*, Intercostal-u. Hodenneuralgie, Presse méd., 1855, No. 37.

—*Dardel*, Deux observations de névrose ano-vésicale opiniâtre, sans cause matér. appréciable, Gaz. méd. de Lyon, 1867.—*Weir Mitchell*, Anal and Perineal Neuralgia, Philadelphia Med. Times, 1873.

General Characters of the Disease.—The neuralgiæ affecting the sensory branches of the sacral plexus (fifth lumbar, and the first five sacral nerves) can, without difficulty, be divided into two groups. The first group contains those forms of neuralgia which occur in the neighborhood of the anus, in the perineum, and in the external generative organs, and are referable to the branches of the pudendo-hæmorrhoidalis; we shall discuss them in the concise account that will hereafter be given of the other forms of neuralgia of the external generative organs. The other group contains the neuralgiæ affecting the posterior femoral cutaneous nerve, and the sciatic, the pain being localized in the posterior surface of the thigh and throughout the whole length of the lower leg and foot (with the exception of the parts supplied by the saphenus nerve). These constitute by far the most important and the most common of the neuralgiæ of the sacral plexus, and are ordinarily included under the general term of *sciatica.* Sciatica and neuralgia of the fifth are the most common and the most important of all the forms of neuralgia. The attacks may be very violent and obstinate, and the treatment may encounter great difficulties. They require, therefore, to be treated of at some length.

Neuralgia ischiadica.—Sciatica.—Malum Cotunnii.—(Hüftweh.)

This neuralgia may affect the greater portion of the posterior surface of the thigh, and a part of the buttock (nervus cutan. fem. poster.); the knee-cap and knee-joint (nervi articulares genu sup. et inf.); the posterior, anterior, and lateral surfaces of the lower leg, and the whole foot with the exception of its internal border (the nervus cutan. crur. post. med., the nervi communicantes peron. et tibial., the nervi peron. superf. et profund., and the nervus tibialis, with its plantar terminal branches). The whole of these nerves are not always coincidentally affected, but for the most part one, or two, or three only. The posterior surface of the thigh, as far as to the middle of the calf, is most frequently the seat of the pain, and then the external surface of the lower leg and foot, and more rarely the sole alone. As in all neuralgiæ, one side only is, as a rule, affected, the right as frequently as the left; occasionally, however, both sides are attacked, though in that case one is usually much more severely affected than the other.

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Etiology.

Predisposition does not here exert so powerful an influence as in many other forms of neuralgia; the greater number of cases of sciatica being occasioned by accidental, more or less external and local causes. Anstie, however, admits even for this neuralgia the influence of premature old age, and incipient bodily weakness, slight signs of which he maintains may be observed in apparently strong persons when they are attacked by sciatica. Such cases are, however, always in a minority, and the nature of the best known accidental causes of sciatica explains of itself why it occurs with such special frequency in strong, robust, hard-working persons, who expose themselves without stint or fear to all external causes of disease. The decided preponderance of male over female patients is in part at least referable to the same cause. According to Valleix the proportion of males to females is 72 : 52; according to Eulenburg, 25 : 7; and according to my own observation, 40 : 10. Its more frequent occurrence in middle life is likewise due to the circumstance that then the struggle for existence leads to the greatest exertions, and to the greatest amount of exposure to external injuries. The effects of cold weather and of various climatic conditions in favoring the occurrence of attacks of the disease may in great part without doubt be referred to the accumulation of atmospheric influences; and again the great frequency of ischiatic neuralgic affections is to some extent referable to the circumstance that the great length and superficial position of the sciatic nerve, and its relations to the pelvis and pelvic organs, render it specially liable to external injuries.

Lastly, it cannot be denied that the nervous diathesis, hysteria, and disorders of menstruation are also predisposing causes.

The accidental causes are far more important than the predisposing, in the etiology of sciatica, and pre-eminent amongst them is *catching cold.* Exposure to a draught of air when the body is heated, wetting of the clothes, sleeping on damp ground or close to a damp wall, have so frequently been known to cause sciatica, that no doubt can be entertained of their activity. In the next place, injuries to the nerve must be mentioned as causes. I have personally had the opportunity of observing the most severe forms of sciatica follow gunshot wounds of the sciatic nerves, in the case of soldiers serving in the last war; I have also known it to follow wounds made in the performance of the operation of venesection, fractures, falls upon the buttock, and long and difficult confinements, especially where the forceps had been used, and the sacral plexus had been injured. *Violent exertions* of the lower extremities are regarded by Anstie as frequent causes of sciatica, especially in those who are predisposed to it. Seligmüller observed the use of the sewing-machine to be a cause in one instance. *Disturbance of the venous circulation*, which probably occasions mechanical irritation of the sacral plexus, not unfrequently induces sciatica, as do also venous stases in the venous plexuses of the pelvis consequent on hemorrhoids, occupations requiring a persistent sitting posture, habitual constipation, and stoppage of the circulation in the portal vein. *Mechanical pressure* may produce sciatica in many ways, whether occasioned by sitting upon hard, uncomfortable seats, or by constipation and accumulation of fæces in the sigmoid flexure, enlargements and dislocations of the uterus, pregnancy, tumors of the pelvis and of the pelvic organs, especially cancer, and ovarian tumors, aneurisms, ischiatic hernia, etc.

Anatomical Changes.

While a variety of changes have been observed in the nerve itself, nothing has yet been discovered which constantly accompanies the neuralgia, so, that for the present, we must regard the anatomical changes as accidental causes of disease. Those that have been observed are neuritis (I have recently observed a fearful case of sciatica, consequent on neuritis, spreading from an obstinate case of inflammation of a tendinous sheath in the lower leg), hyperæmia, œdema, and exudation in the neurilemma, deposit of tubercle and ossification in the neurilemma, ectasis of the veins, tumors, cicatricial neuromata, etc.

Diseases of the vertebræ (spondylitis, caries, cancer, etc.), are frequently accompanied by severe symptoms of sciatica; so, also, diseases of the *central nervous system*, especially of the spinal cord, and, more rarely, of the brain, such as meningitis spinalis, myelitis, and, above all, tabes dorsalis are often coupled with excentric neuralgic pains in the sciatic nerve (usually in both). Sciatica has been observed as a sequela of typhoid fever, and as a result of syphilis. All authors consider it doubtful whether it can be induced by malarial infection, as a typical example of sciatica is extremely rare in periodic fevers. Sciatica, as I am informed by experienced friends, is not unfrequent in diabetes, whilst Braun states conversely[1] that he has often observed diabetes as a symptom of sciatica. There are, lastly, many conditions that may occasionally induce sciatica, without their importance being fully recognized, as *e. g.* suppression of the perspiration from the feet, healed eruptions, lead poisoning, reflex actions from the digestive organs, etc. Their efficiency as causes, however, must be established by future research.

Symptoms.

The disease commences with certain premonitory symptoms, such as a dull, painful dragging, and stiffness, or feelings of formication, or a sensation of fluid trickling over the skin, or of cold, or heat in the affected extremities. The symptoms proper of the disease, sooner or later, in different cases, now begin to make their appearance; painful, lightning-like pains are experienced, which gradually rise in intensity, and lead to the outbreak of a violent paroxysm. Such attacks rarely occur quite suddenly, and without any warning. The pain in the attack is remarkably violent, and of a tearing, lancinating character, traversing the leg in the direction of the nerve trunks, with constantly increasing violence and frequency. These pains proceed from one or more fixed points, and extend usually in a descending, but occasionally in an ascending direction, and sometimes in several directions. The pain often rages at a definite point with violent lightning-like shocks, then changes its seat and becomes excruciating at another point, and, finally, returns to its former situation. It is ordinarily seated in the skin, but often also in the deeper tissues in and between the muscles, not unfrequently, also, in the bones. The patient is often able, from the position of the pain, to trace out with his finger with surprising accuracy the anatomical disposition of the main trunks of the nerve, and the lines which connect the most painful spots often correspond exactly with the course of the nerves.

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[1] In his Balneotherapie, 3. Auflage, p. 411.

The intervals between the paroxysms of lancinating pains are, as a rule, by no means entirely free from pain. A dull feeling of tension and uneasiness is perceived in the affected parts, which seldom disappears entirely. In general, therefore, there is only a remission of the pain. The exacerbations and the times when the attacks are frequent occur chiefly in the evening and night, the violent pains not unfrequently preventing sleep for many hours. Cases occasionally occur, however, in which the nights are peaceful, but the days bring back the pain. Many circumstances are capable of augmenting the pain and inducing paroxysms. This is especially the case with movements of the affected leg, a change of position in bed alone being sufficient for the purpose; and the pain is certain to be brought on by strong and persistent movement. Nevertheless patients are occasionally met with who find relief in walking about, and who cannot remain in bed. Simple contact of the clothes with the skin, cold air, washing the legs, coughing, sneezing, sudden bending of the body, and straining at stool, are all common exciting causes of an attack. These various circumstances constrain most persons to be very cautious in the use of the leg, and in all their movements; hence the peculiar and very characteristic position in which the leg is kept, with all the joints slightly bent, and as far as possible in an immovable state, in order that every jar or pressure on the sciatic nerve may be avoided.

The area affected with pain varies considerably in extent in different instances, and, as different branches are chiefly affected at different times, all possible combinations occur. Most commonly the patients complain of pain in the posterior surface of the thigh, the pain commencing in the neighborhood of the sciatic foramen, and extending to the popliteal space and calf of the leg; next to this the region of distribution of the peroneus (anterior and external surface of the leg and dorsum of the foot) is the part that most frequently suffers. Much more rarely the region of distribution of the tibialis is affected, though cases have been observed where the pain was confined to the sole of the foot (neuralgia plantaris). Not unfrequently the whole area of distribution of the nerve is affected at the same time, and the pain occurs with special violence first in one and then in another nerve. Then, as a rule, the posterior roots of the sacral nerves are also implicated, and the patient complains of violent pains in the sacrum and in the loins.

Painful points are rarely absent in sciatica, yet there are undoubtedly cases in which it is impossible, even with the most careful examination, to discover the presence of any such points. Valleix considers a point close to the sacrum, in the immediate vicinity of the posterior superior spine of the ileum, to be the most constant one, but I find a point corresponding to the point of emergence of the sciatic nerve from the pelvis to be the most frequently sensitive to pressure; another is found at the lower border of the gluteus, at the spot where the posterior cutaneous nerve emerges. In the popliteal space there is a painful line corresponding to the course of the tibial nerve, and a painful point over the head of the fibula; there is also a point behind the outer ankle bone and another behind the internal malleolus. There are several points on the dorsum of the foot, and not unfrequently some inconstant ones on the posterior surface of the thigh and on the calf of the leg, at the points where the cutaneous branches divide or where they perforate the fascia. Not unfrequently the sacral plexus itself can be reached by an examination *per anum* or *per vaginam*, and will then be found to be painful on pressure.

Except in neuralgia of the fifth, the concomitant phenomena have been observed with greater care, and are more prominent in sciatica than in any other form of disease.

Radiation of the pain to the sciatic nerve of the opposite side is exceedingly common during the height of the attack, and occasionally it shoots into the lumbar nerves and their branches, or even into more remote nerve regions. Abnormal sensations, such as formication, numbness, furriness, cold, are often felt in the affected extremity. There is often, also, some amount of hyperæsthesia or anæsthesia in the area of the skin affected and in the adjoining parts, as Nothnagel has particularly noticed (see above). Not unfrequently there is well-marked partial anæsthesia localized in different parts of the skin.

Amongst the *motor symptoms*, the limping gait and the peculiar mode of carrying the leg, which has been already mentioned, are remarkable. Cramp in various degrees is not uncommon, sometimes consisting of only slight fibrillar contractions, with moderately painful tension of the muscles, at others rising to violent tremors and the most frightful convulsions, which increase the sufferings of the patient to an inconceivable extent. These symptoms are the result of direct or reflex irritation. Of more serious importance are the paretic and paralytic conditions which not unfrequently supervene. Slight paresis, weakness, and stiffness are found in almost all cases; but if there be more marked paresis or absolute paralysis of any groups of muscles, it may be inferred that serious lesions of the nerves are present.

The *vaso-motor trophic disturbances* in sciatica have already been discussed in the general part. Paleness and coldness of the skin, with actual chilly sensations and numbness, are of common occurrence; in other cases redness and heat of the skin, with increased secretion of sweat occur, or these two states may alternate. The most common trophic disturbance is simple moderate atrophy of the muscles, though a high degree of atrophy is observed only when there is at the same time well-marked paralysis. Graves states that he has seen hypertrophy of the muscles; Anstie has noticed increased growth of hair; while zoster has seldom been observed. Flies noticed in one instance an eruption of numerous boils. Braun[1] has observed in several instances the occurrence of sugar in the urine as a symptom of sciatica.

General disturbance of the health, such as is indicated by febrile symptoms, scarcely ever accompanies sciatica, yet the violence of the pain may, by preventing sleep and interfering with the appetite and digestion, gradually cause complete exhaustion, and, of course, the various lesions that occasion the sciatica, as cancer, caries of the vertebræ, tabes, etc., may lead to general disorder of the system of the most serious nature, which cannot, however, be attributed to the sciatica.

Eulenburg[1] has made some remarkable statements, at least in reference to the motor fibres, upon the *electric excitability* of the sciatic nerve in sciatica; he observed repeated qualitative changes in the normal law of contraction, even to its complete reversal, besides quantitative increase or diminution of the galvanic excitability. I have not hitherto observed anything of this kind, except when a serious degree of paralysis and muscular atrophy has been present, in which case these reactions have quite a different significance. The sensibility of the skin to faradic currents is frequently, in accordance with the degree of anæsthesia present, somewhat diminished. A minute investigation of these relations, and a careful report of the observations made, is a desideratum.

Course, Duration, Terminations.

The disease, as above stated, usually commences gradually and rises to a certain intensity, at which it remains with some variations for a longer or shorter time. As a rule, sciatica lasts for several weeks. It is rare to effect a cure in a few days, for too frequently the disease persists for months. The attacks usually occur somewhat irregularly, and it is rare to observe a well-marked periodic character about them. As a rule, great variations occur in the intensity of the symptoms, and these variations appear as a result of mental emotions, physical exertion, atmospheric variations, low barometric pressure, etc. Improvement takes place quite gradually, and usually with many relapses. Even when recovery is complete, the patient suffers for a long time from the feeling of a diseased and weak leg. It remains stiff, awkward, easily tired, and the pain recurs upon very slight provocation ; in fact, relapses are very common.

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[1] Balneotherapie.

[2] Loc. cit., p. 154.

The *duration* of the disease varies from a few days to thirty years, but in the so-called idiopathic cases the usual duration is from two to eight weeks.

The disease usually terminates in *recovery*, though unfortunately it is often only incomplete, the neuralgia indeed disappearing, but the leg remaining stiff and weak, with atrophy, disturbance of the sensibility, and disposition to relapses ; indeed the traces of an attack are often perceptible for years. Bad results are usually dependent on the nature of the primary disease, and must not be attributed to the neuralgia.

Diagnosis.

The diagnosis of sciatica is not unfrequently surrounded with great and sometimes insuperable difficulties ; this is especially apt to be the case in very fat persons, in women, and in uncultivated people, who can give only an imperfect account of their sensations, and in whom the results of examinations are uncertain. The affection is most likely to be mistaken for muscular rheumatism of the thigh or leg, and although the characteristic diagnostic sign given by Valleix, that in rheumatism the patient indicates the seat of the pain with his whole hand, whilst in neuralgia he points to it with his finger, does not hold good in all cases, yet a correct diagnosis may in general easily be deduced by attention to the already enumerated signs of rheumatism, viz., its localization, the pain produced in the performance of definite movements, easy curability, etc.

The diagnosis of sciatica from hip-joint disease is also very difficult as well as important. It is especially in the insidious and slowly developing forms of coxitis and coxarthrocacia, which run on for a time without fever, without perceptible deformity of the pelvis, and without any marked local sensibility, that errors are likely to be made. Positive conclusions can only be arrived at by the exercise of the greatest care in the examination of the patient. The position and mode of carrying the leg, the situation of the points that are painful on pressure, the absence or presence of pain when the head of the femur is pressed against the acetabulum, the elongation or shortening of the leg, the configuration of the lower part of the back, the paroxysmal character of the pain, its distribution, and the conditions of the general health, are all circumstances that must be carefully considered in framing the diagnosis, which becomes still more difficult in those cases of nervous coxalgia (hysterical articular neurosis) that have been described by Brodie, Esmarch, Wernher, Stromeyer, and others. The precise localization of the pain in the hip-joint, and the absence of inflammatory symptoms must constitute the principal data on which the diagnosis must rest.

The mistake of confounding sciatica with psoitis can only be fallen into from sheer heedlessness.

The diagnosis of the *seat* of the neuralgia, in regard to the part of the nerves affected, is of great importance ; that is to say, whether the disease is seated in the periphery, in the plexus, in the roots, or in the central parts of the nervous system. Careful attention to all the circumstances will, in general, render it practicable to make a correct diagnosis upon this point. Thus, the disease will probably be of peripheral origin when it is localized in particular branches, and there are well-marked coincident motor and vaso-motor disturbances ; it will probably be in the plexus when the pain has a wide distribution, and when certain indications are obtained from an examination per rectum or per vaginam ; again, in favor of its originating in the roots or in the centres, is the extension of the pain to the posterior branches of the sacral plexus, its well-marked lancinating character and its localization in the bones, and evidence of other symptoms of central disease. The proof of a definite locally acting cause is of great value in the diagnosis of these several forms. The diagnosis of the causes occasioning sciatica must be made on general principles, and is obviously of great importance in determining the treatment to be adopted.

Prognosis.

The prognosis is less favorable in sciatica than in crural neuralgia, yet it is better than in most other forms of neuralgia, in which the symptoms are equally violent. It is primarily influenced by the cause of the disease ; if it originate in serious and incurable lesions, the prognosis is of course very bad. Recent rheumatic and idiopathic cases almost always admit of a favorable prognosis being given, and it may be predicted with some certainty that recovery will take place in a very few weeks. Slowly developing cases, associated with anæsthesia, paralysis, and atrophy, are usually obstinate.

The longer the duration of the disease the worse is the prognosis, and it is worse also as the age of the patient advances. The liability to relapses should also always be borne in mind.

Treatment.

In this, as in all other diseases, the causal indications of treatment must first be attended to. This can in many cases be effected by operative treatment, as by removal of tumors, coaptation of fractures, resection of cicatrices, and removal of foreign bodies. In other instances, where venous stases, constipation, etc., are present, good results may be obtained from the employment of purgatives, saline waters—such as those of Kissingen, Homburg, Marienbad, Tarasp, etc.—and from leeches applied to the anus or perinæum. When the disease arises from wounds of the gluteal region, antiphlogistic means may be employed, as cold, abstraction of blood, rest, and compressing bandages. Plouviez recommends the use of a pressure-bandage over the whole extremity in traumatic neuritis as well as in sciatica due to cicatricial neuroma. When the affection is of central origin, the appropriate treatment for the particular lesion present must be adopted, and so on. Recent rheumatic cases, occurring in robust patients, constitute by far the majority of all the cases that present themselves for treatment, and in these energetic diaphoresis and derivation by the skin are the chief indications ; indeed, it is not uncommon to effect a perfect cure of recent cases in a few days by a thorough sweating, as in a vapor or Turkish bath, in connection with the application of wet cups or some strong cutaneous irritant, (such as blisters) and absolute rest. In chronic rheumatic cases, the iodide of potassium, and the indifferent thermal baths, prove serviceable.

The number of specific remedies for sciatica is enormous, and only the more important can here be mentioned. In slight cases, cutaneous irritants suffice to cure, and consequently occupy a prominent place amongst the remedies for sciatica. Nothing is more effective than the application of flying blisters, originally introduced by Cotugno, and strongly recommended by Valleix. These, which may be of the size of a visiting card or the palm of the hand, may be applied at intervals of two or three days over the parts that are especially painful, and, when the blister is fully formed, the sore surface may be allowed to heal beneath a simple bandage. The results are generally brilliant, but in many instances the pain is cured only over the superficies actually covered by the blisters. Anstie recommends the application of the blisters to the sacrum (blistering of the posterior branches) as extremely effective, but admits at the same time that they often fail to relieve the pain until they are applied over the nerves themselves. The keeping open of the blisters by means of irritating ointments is not very advisable, as the pain usually returns after a short time. Less is to be expected from the slighter cutaneous irritants than from vesicants, but if it be determined to apply them, sinapisms, camphorated liniment, irritating ointments, oil of turpentine, either alone or in combination with croton oil, mezereum, and the fresh leaves of Ranunculus acris (an old and recently revived popular remedy) may be employed. Betz recommends, as a very effective remedy, an application of a plaster, composed of from fifteen to twenty-three grains of nitrate of silver mixed with half an ounce of Saffron plaster,[1] over the painful parts. This may be allowed to remain on till it falls off. It produces strong burning and eczema, and, as it is claimed, causes the pain to cease in the course of one or two days.

In the more serious cases, where it is desirable to use more energetic measures, the *actual cautery* is a sovereign remedy, and its application is often followed by brilliant results. It is usually recommended to cauterize the skin superficially in transverse lines (transcurrent cauterization—Valleix, Jobert); while the plan of cauterizing more deeply (moxæ) and thus inducing suppuration for some time afterwards, is less to be recommended. The plan of establishing issues is open to the same objections. The actual cautery is best applied immediately over the affected nerves, but it has been tried in remote parts, as between the toes and on the dorsum of the foot. The palliative effect of the application of a thin, blunt cauterizer to the external ear is very remarkable, and has been corroborated by the best observers. Our ignorance of its mode of action is no reason why it should be held up to ridicule.

In most cases, and especially in those where, from the nature of the primary cause of the disease, no complete recovery can be anticipated, it is impossible to dispense with the use of *narcotics*. They may often be advantageously used to aid the action of the derivative measures employed; in many cases they constitute the only satisfactory palliative means we possess. Nothing remains to be added here to what has already been given in the general part.[2] The subcutaneous injections of morphia in doses of from one-sixth to nine-tenths of a grain occupy the first position in point of efficiency. They are best made in the neighborhood of the sciatic foramen, or over one or other of the painful points. It is often requisite to follow out the several branches of the nerves with the injection, in order to suppress the pain in all parts, and hence it is frequently advantageous to make several injections coincidently at different points, and in double sciatica in both legs. If morphia prove ineffective, atropine may be tried; the other narcotics are, for the most part, of little value. Their employment in the form of external applications, as in ointments containing veratria and opium, or aconitine, extracts of belladonna or hyoscyamus, etc., is deserving of little confidence. The method frequently recommended by Trousseau deserves mention: to an issue on the buttock, two or three pilules are applied daily, each of which contains three-quarters of a grain of extract of opium and the same quantity of extract of belladonna, made up with gum tragacanth. The *anæsthetics*—chloroform, ether, etc. —may be used as liniments, and especially in the form of clysters, in sciatica, the palliative action of the latter, in particular, being very marked.

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[1] The "Saffron plaster," *emplastrum oxycrocum* of the German Pharmacopœia, is compounded of yellow wax, resin, Burgundy pitch, ammoniac, galbanum, turpentine, mastic, myrrh, olibanum and saffron.

[2] See above, p. 81.

A knowledge of the extreme value of *electricity* as a curative agent in sciatica is of recent date, but has been carefully investigated. It deserves to be tried in all severe cases, and especially —where it is found practicable—in slight and recent cases; the results in most instances being excellent. This is certainly true, to a less degree, of the faradic current, which only occasionally affords good results. It may be applied in the form of the electric brush or the moxa, or with moist electrodes, which occasion a strong secondary current in the nerve trunks. The action of the galvanic current is much more energetic and effective in sciatica, and from the concordant results of electro-therapeutists it appears to be equally successful in recent and in chronic cases, it being presupposed, of course, that the affection does not depend upon incurable organic disease.[1] I have effected a cure after a few sittings in several cases,[2] some of which were chronic, though in other instances a long-continued conrse of treatment of this kind was requisite. The most convenient method is the application of descending currents with stabile electrodes, the anode being placed upon the sciatic foramen, or, better still, upon the sacrum, and the cathode upon the specially painful parts. It is often advantageous to adopt Remak's plan, and to bring separate portions of the nerves, from six to eight inches in length, successively under the influence of the current, beginning at the sacrum, and passing down to the foot. A few muscular contractions are then excited by closure of the current, which readily removes the feelings of stiffness and weight in the leg. Another method, which has already been employed by Remak under the name of circular current, consists in the stabile application of the anode upon the trunk of the nerve and upon the painful points. Strong currents must always be employed with broad electrodes, on account of the depth at which the nerve lies. The duration of the application of the current should be from five to ten minutes daily, or every second or third day. The rule "treatment in loco morbi" must obviously here be put in force, and in certain cases, therefore, the neighborhood of the sacrum and the vertebral column must be included in the treatment. It is also not unfrequently necessary that in applying the current to the painful nerves, each individual painful twig should be subjected to its influence. Special symptoms, moreover, like anæsthesia, paralysis, etc., require special modifications of treatment. Benedict has recommended that in particularly severe cases, one electrode should be introduced into the rectum, and the other placed upon the sacrum, in order that the current should be applied as directly as possible to the seat of the disease. The results thus obtained in one case were very satisfactory. The plan repeatedly recommended by Ciniselli,[1] of the continuous application of a single galvanic element—one zinc and one copper plate connected by a wire—to the affected limb, is worthy of trial. Such plates can be adapted to any part of the skin, and may be worn for hours or for days together. The results are in many instances remarkable.

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[1] See the various Manuals on Electrotherapeutics.

[2] See also Deutsches Archiv f. klin. Med., Bd. III., p. 342.

When all these means fail, recourse may be had to *specifics.* The number of these is very great, and a few may without difficulty be selected, the use of which has been followed by good results. Foremost amongst these is rectified oil of turpentine. This has been very generally recognized as an effective remedy for 150 years, though the precise indications for its exhibition have never yet been laid down. This much is certain, that whilst in some cases it proves extremely serviceable, in others it is useless. It has been especially recommended by Martinet, Recamier, and others, and may be prescribed in various forms, as, for example, combined with honey in the proportion of one part of oil of turpentine to fifteen of honey, two or three tablespoonfuls being taken daily. An agreeable mode of taking it is in the form of the gelatine capsules, each containing about fifteen grains, and of which from three to twelve may be taken daily at meal times. The good results are generally perceptible in the course of a few days. After turpentine, arsenic is celebrated for its efficacy. From quinine little, as a rule, is to be expected ; it has been given without effect, even in typical cases. Iodide of potassium is very useful in many cases. Mercurial preparations, especially corrosive sublimate, ergot, bromide of potassium, cod-liver oil, need only be mentioned, but it may be added that the ancients regarded music as a remedy for sciatica —certainly without any conception of the achievements of the most modern school of music.

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[1] See Virchow-Hirsch, Jahresbericht über Electrotherapie, 1867 and 1872.

While but little dependence can be placed upon the remedies we have just enumerated, the various kinds of baths will be found to afford additional and very important means of cure for sciatica. It may, however, first be observed that the persistent application of heat has been found useful. Plouviez recommends the application of poultices (at a temperature of from 104° to 122° Fahr.) to the affected leg. The indifferent thermal baths enjoy the greatest repute, and there can be no doubt that many severe and obstinate forms of sciatica have been cured at Teplitz, Gastein, Wildbad, Ragatz, Wiesbaden, and Baden-Baden. We possess, however, no precise indications in regard to their use, but chronic rheumatic forms are probably the most likely to be benefited. Good results have also been obtained from mud baths. The favorable influence of the cold-water treatment, vigorously carried out, is indubitable, even when the cases are very severe and obstinate. The warning of Romberg against this method of cure has been found to be uncalled for. Sea-water baths have also been found serviceable. It need scarcely be added that all these forms of baths must be selected in accordance with the special circumstances of the case.

Surgical operations (at least on the main nerve trunks) can only be undertaken in cases that are perfectly desperate; they may, however, be performed on the smaller sensory branches without fear, but resections of the sciatic nerve itself, or of its two terminal branches, should be carefully considered before being undertaken, on account of the subsequent paralysis, which is generally incurable. Sufficiently strong reasons for the operation may, it is true, be present, and it has been performed with success. The decision must rest on general grounds. Patruban has recently published a case of obstinate sciatica, in which he adopted, with good results, the plan suggested by Nussbaum of cutting down upon and stretching the sciatic nerve; in this case the power of voluntary control over the movements of the limb remained unaffected. Further observations must demonstrate the applicability of this bold operation.

Little need be said in regard to the dietetic treatment of those who are suffering from sciatica, as this will naturally depend upon the conditions peculiar to each individual case. In all cases, however, the most important accessory means of aiding recovery is rest in the recumbent position. Care should also be taken that the bowels are evacuated regularly.

Neuralgia Pudendo-hæmorrhoidalis, and Neuralgiæ of the External Generative Organs generally.

The rarity and unimportance of the neuralgiæ included under the head of neuralgia pudendo-hemorrhoidalis render it unnecessary to enter into any minute details in regard to their symptoms. The principal region of distribution of this nerve is in the external genital organs. These parts, however, receive sensory nerves from so many different sources that it is quite impracticable to consider the neuralgiæ of the several nerve regions separately. Moreover, the sympathetic plexus of nerves plays an essential though not very precisely limited part in the innervation of the external generative organs, and the difficulty of giving an exact account of the affection is thus considerably increased. It will probably therefore be sufficient to consider these rare affections very briefly, in order to afford at least some guidance to the physician who may happen to meet with them. The pathology and treatment of these neuralgiæ are still extremely defective.

The following are the sensory nerves supplying the external generative organs and neighboring parts: the ilio-inguinal and external spermatic nerves, supplying the mons veneris; the external spermatic, the posterior scrotal nerves (branches of the pudendo-hæmorrhoidalis), and the inferior long pudendal (a branch of the posterior cutaneus femoralis) supplying the scrotum and labia majora; the dorsalis penis and clitoridis (a branch of the pudendo-hæmorrhoidalis), and the branches from the plexus cavernosus sympathici, supplying the penis and the clitoris; the dorsalis penis and the posterior scrotal nerves, supplying the urethra; sympathetic branches from the sympathetic plexuses which surround the vas deferens and spermatic cord, supplying the vas deferens and the testes; and, lastly, branches from the external hæmorrhoidal, and from the perineal nerve, supplying the perinæum and the neighborhood of the anus.

It thus appears that the external genitals may participate in neuralgiæ affecting a variety of nerves, as in cases of lumbo-abdominal neuralgia, in sciatica, and in neuralgia of the pudendo-hæmorrhoidal nerve. Little is, however, known in regard to this point. It is only in lumbo-abdominal neuralgia that this participation of the external genitals in the pain constitutes so prominent a symptom as to receive notice. This also is a subject requiring further investigation.

Isolated neuralgiæ of the genitals, analogous to mastodynia in intercostal neuralgiæ, are, however, sometimes encountered. Several nerve regions are usually affected, and the determination of the particular nerves implicated is very difficult. The following forms, founded on reports of cases, may perhaps be distinguished:

Neuralgia of the penis and glans penis. This has its seat in the glans penis, and extends to the root of the organ. The pain may be very violent, lancinating, and burning, and is usually increased in passing water, or by sexual intercourse, and is frequently accompanied by priapism and frequent ejaculations; it is often unilateral. A case recorded by Liegey (loc. cit.) belongs to this form, and I have seen a similar case, which lasted for several years, and resisted all treatment.

Neuralgia scrotalis (labialis) is one of the commonest symptoms in lumbo-abdominal neuralgia, and presents the same characteristics. It is termed neuralgia ileo-scrotalis by Chaussier. The scrotum or labium majus is often extremely tender to the touch.

Neuralgia urethralis. In this affection the patient experiences burning pain in the urethra, which is especially apt to occur when the urine is voided drop by drop, and is usually accompanied by increased desire to urinate. The introduction of a catheter is both difficult and very painful. It is often an early symptom of tabes dorsalis, but it may also result, according to Eisenmann, from catching cold or from morbid conditions of the urine.

Neuralgia spermatica. This is characterized by violent intermitting pain in the testis and epididymis, which radiates along the spermatic cord and often down upon the thigh. At the same time these parts become extremely sensitive to pressure (Cooper's "irritable testis") and are liable to periodic enlargement. The attacks are not unfrequently accompanied by general malaise and vomiting. The affection is usually limited to one side only, and occurs chiefly in young persons. Opinions are divided in regard to the seat of the affection; Valleix and d'Axthrey consider it to be a form of lumbo-abdominal neuralgia, whilst most German authors (Romberg, Hasse, and Eulenburg) regard it as a sympathetic neuralgia. The latter opinion seems to be probable, since the testis receives its nerve-supply almost exclusively from the sympathetic, and since also, from the very mode of its development in the fœtus, it can hardly obtain any branches from the scrotal nerves (see the Diseases of the Sympathetic Nerve).

Under the head of *neuralgia ano-vesicalis*, Dardel has described certain morbid sensations in the region of the coccyx, which are associated with hyperæsthesia (or sometimes with anæsthesia) of the skin of the perineal region, with cramp-like contraction of the sphincter ani and of the bladder, and with difficulty of micturition. Weir Mitchell has described a similar affection under the name of "anal and perineal neuralgia."[1] It occurs especially in patients suffering from tabes and in onanists, frequently also after coitus.

The treatment of all these affections must be conducted on general principles. The milder forms usually yield to the ordinary treatment for neuralgia, whilst the severer forms often prove intractable under every plan of treatment. Narcotics are generally employed both internally and externally, but best according to the hypodermic method. Specifics (arsenic, quinine, bromide of potassium, oil of turpentine, etc.) cannot, in general, be dispensed with. We do not possess a sufficient number of data to enable us to judge of the value of electric treatment. Operative proceedings (and even castration) have now and then been tried, in neuralgia spermatica, and occasionally with success.

g. *Neuralgia of the Coccygeal Plexus—Coccygodynia.*

The coccygeal nerves are distributed in the skin over the coccyx and in the adjoining soft parts. Whether they can be the seat of a true neuralgia, appears still to be doubtful, though a certain number of the cases described by Simpson[2] under the term "coccyodynia" are probably of a neuralgic character. The chief symptom of this *coccygodynia* is pain felt in the region of the coccyx in sitting and walking, and frequently also in micturition and defecation, if at these times the patient strain hard. Pressure upon the coccyx and movement of it with the finger also induce pain. The affection may last for many years, and is almost always observed in women, in whom it occurs either in consequence of injury to the coccyx, as by a fall or during a severe labor, or after catching cold; it frequently also seems to originate spontaneously. The probable cause of the pain is the tension of the muscles inserted into the coccyx, owing to the fact that the bone itself or its fibrous investment is the seat of inflammatory irritation (or perhaps to the fact that the coccygeal nerve is neuralgically affected ?).

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[1] Philadelphia Med. Times, 1873.

[2] Med. Times and Gazette, July, 1859.

In cases where the neuralgic character of the disease is well marked, anti-neuralgic treatment may be successful. Thus Seeligmuller[1] cured a case which had lasted for many years by faradization, but this mode of treatment, as a rule, is futile, and a cure can only be looked for through operative means. This consists in completely separating the coccyx from all fibrous and muscular fibres that are connected with it, which may be accomplished by subcutaneous section with a tenotomy knife, without much pain or hemorrhage, and has proved successful in many cases. If, however, this fails, the coccyx must be extirpated.

APPENDIX.

Articular Neuralgia.—Hysterical Affections of the Joints.—Neuroses of the Joints.

B. Brodie, Diseases of the Joints, 2d edit. London, 1822.—*Brodie*, Lectures Illustrative of certain Local Nervous Affections, 1837. Translated into German by Kürschner. Marburg, 1838.—*Stromeyer*, Handbuch der Chirurgie. Freiburg 1844.—*Volkmann*, in Pitha and Billroth's Chirurgie Band II., Heft 2, p. 678, 1872.—*Esmarch*, über Gelenkneurosen, 1872.—*Wernher*, über Nervöse Coxalgie, Dtsche Zeitschr. f. Chir. I., 1872.—*Stromeyer*, Erfahrungen über Localneurosen, Hannover, 1873.—*O. Berger*, Zur Lehre v. d. Gelenkneuralgien, Berl. klin. Woch., 1873, Nos. 22–24. Records of Cases will be found in *Hirsch*, Spinalneurosen, 1843; *Barwell*, Diseases of the Joints, 1865; *Marion Sims*, Clinical Notes on Uterine Surgery, New York, 1871; *Skey*, On Hysteria, 1867; *Benedict*, Electrotherapie, 1868.

Brodie was the first to call attention to certain painful affections of the joints which closely resembled neuralgia, and in which both careful observation and the course of the disease rendered it almost perfectly certain that there were no organic changes in the joint. This celebrated English surgeon gave it as his opinion that at least four-fifths of all the articular affections occurring among women of the higher classes are of this nature—a statement that has recently been corroborated by Esmarch. As this affection occurs chiefly in association with well-marked hysterical phenomena, it has been customary to designate it by the term "hysterical joint disease;" but this term not being applicable to all cases, Esmarch has proposed the more general term "articular neurosis," whilst Berger has decided in favor of the term "articular neuralgia" as the one which best expresses the essential features of the disease.

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[1] Neuropathische Beobachtungen. Halle, 1873, p. 25.

It cannot be denied that this affection presents very marked analogies to true neuralgia, and that it has been regarded as such by most authors; quite recently, too, Esmarch has expressed himself in favor of this view, whilst Berger has adduced some very strong evidence showing that the affection is in reality a neuralgia. Nevertheless, the limitation of the pain to special nerves, which is so characteristic a feature of other neuralgiæ, is here absent, for most joints receive their nerve supply from different sources (the hip-joint, for example, being supplied by the crural, obturator, and sciatic nerves, and the knee-joint by the crural, obturator, peroneal, and tibial nerves). On the other hand, the functional unity of the sensory nerves affected may explain the circumstance of their being coincidently diseased, especially in those individuals who are predisposed to neuralgia; and besides, it has not as yet been demonstrated that, in articular neuralgia, all the nerves distributed to the particular joint affected are implicated.

Articular neuralgia has received but little attention in Germany, and one would easily be tempted to accept as true the statement that it is frequent in England, but rare in Germany, were it not that both Esmarch and Stromeyer have adduced many examples from their own experience, which corroborate the statements of Brodie in regard to this affection. The great practical importance of the subject—for a misapprehension of the nature of the disease may be attended with serious consequences to the patient—will justify the introduction of a short sketch of articular neuralgia in this place.

Etiology.

Women exhibit a decided predisposition to the disease, the largest contingent of cases being supplied by young ladies in the better ranks of society, who, as a result of their irrational mode of life—party-going and attendance upon nocturnal amusements, as well as improper diet, insufficient physical exertion, coddling, and over-indulgence—get their nervous systems into a state of irritable weakness which is a very fruitful source of hysterical symptoms and hysteria. At all events hysteria—however it may originate—strongly predisposes to articular neuralgia. All depressing and deep-seated emotions, as well as large or frequently repeated losses of blood, appear to act in the same way. It must not, however, be overlooked, that perfectly healthy and robust patients, maid servants, etc., and even occasionally men, become the subjects of this, as they may of other forms of neuralgia.

Amongst the *exciting causes* of the disease, mechanical and traumatic agencies occupy the foremost place ; slight contusions and distortions of the joints, such as frequently occur in falling or upon being struck while dancing, or skating, or in mountain climbing, may, after the immediate effects of the injury have subsided, leave articular neuralgia behind them, especially if an aimless, varying, and too debilitating treatment have been adopted. Violent emotions, fright, fear, etc., not unfrequently cause articular neuralgia. Anxiety in regard to some relative who is suffering from serious articular disease, and the dread of falling a victim to the same disease, may be the cause of articular neuralgia. Catching cold has sometimes proved a cause. Irritation of the abdominal viscera from some article of food that has been taken, and all sorts of pathological conditions have been recorded as causes, the urinary and sexual organs being particularly fruitful in this respect (see cases by Esmarch, Stromeyer, Sims, and others). The numerous connections that exist between the pelvic plexuses of the sympathetic and the sacral and lumbar plexuses, perhaps afford the key to this fact. Direct mechanical irritation of the nerves supplying the joint has likewise been observed as a cause; thus in one case of neuralgia of the knee-joint an aneurism was evidently the cause. Lastly, articular neuralgiæ frequently occur after acute diseases, and Berger has noticed that it may constitute a symptom of certain diseases of the central nervous system (as of hemiplegia, tabes, etc.).

Symptoms.

The disease usually comes on without any premonitory symptoms, and may supervene quite suddenly. Usually, however, it develops gradually from slight beginnings to the more severe grades.

The principal symptom is more or less acute pain, which is described as dragging, tearing, stabbing, etc., in character, and is not always limited to the affected joint, but radiates from it in various directions. Thus, for example, in articular neuralgia of the hip-joint the pain extends downwards to the knee, in neuralgia of the knee-joint, upwards and downwards along the different nerves implicated. The pain occurs chiefly in paroxysms, presenting very well-marked exacerbations and remissions; it is most violent in the evening, and least so in the morning. As a rule the sleep is not disturbed by it, while just the opposite is true of the pain in inflammation of the joints. When the patient has once fallen asleep, he sleeps through the night. Diversion of the mind to other objects, and in some cases walking about diminishes the pain. The pain is often augmented during the catamenial period, but it often also disappears at this time.

Objective examination of the affected joint yields very little information. It is not swollen, and is often seen less in girth than the opposite one; its temperature is not increased, and there is no shortening of the limb; on the other hand, there are various symptoms that we are accustomed to meet with in neuralgia. In the first place, the joint is extremely sensitive and tender, and especially so at certain points; these are the painful points. Such points are found over the hip-joint, between the trochanter major and the tuberosity of the ischium, near the anterior superior spinous process of the ilium, over the knee-joint (along the inner border of the patella and over the internal condyle), in the popliteal region over the head of the fibula, over the ankle-joint (behind the outer and inner ankle bones), over the shoulder-joint (over the brachial plexus above and below the clavicles and in the axilla), over the elbow-joint (over the external condyle and head of the radius), in the wrist-joint (over the styloid process of the ulna), over the vertebral joints and over the spinous processes (and here it should be mentioned that it is probable, though not easy to demonstrate positively, that some of the cases described as cases of "spinal irritation," belong to the category of articular neuralgia).

In addition to the painful points, there is also usually a very well-marked diffused hyperalgesia of the skin covering the joint, and it is a special characteristic of the disease and one of diagnostic importance that in such patients slight contact of the skin, or the raising of a fold of skin, is much more painful than firm pressure over the joint itself; in accordance with this also is the fact that pressing the two surfaces of the articular extremities against each other produces comparatively little pain.

Anæsthesia of the surrounding skin is sometimes noticed, though rarely (Boddært, Berger). Berger has also observed paræsthesiæ (sensation of burning, formication).

The most common *concomitant motor phenomena* are cramp-like or convulsive movements of various kinds, viz. spasmodic muscular contractions, which cause the limbs to assume unnatural positions, and which may even increase to such a degree as to cause persistent contractions (such conditions are most frequently met with in neuralgia of the hip-joint). In other cases quivering and tremors as well as convulsions may occur. A marked feeling of debility is often present, but rarely actual paralysis. Wernher, however, has described a series of well-marked paralytic conditions (and cramps) as occurring in nervous coxalgia, though some of his cases appear to have been slight forms of true inflammatory disease of the hip-joint.

Vaso-motor disturbances are very common, and may occasionally lead to a false diagnosis. The rapid and not unfrequently regular alternation of congestion, redness, and heat, on the one hand, and anæmia, pallor and coldness of the skin about the joint, on the other, is a characteristic feature of the disease. A considerable degree of turgescence of the skin, amounting even to doughy swelling, may be present, and Brodie describes the occasional occurrence of a large urticaria-like wheal (Quaddel) over the whole joint, in which he sought in vain for pus.

Amongst *trophic disturbances* wasting of the corresponding limb, due to atrophy of the muscles moving the joint, is alone perceptible. Wernher describes an arrest of growth of the whole extremity in young persons, and refers it to the implication of trophic nerves.

The functional disturbances of the affected joint are always well marked, the most prominent being immobility and weakness. The patient avoids moving the joint as much as possible on account of pain, and keeps the limb usually fixed in the extended position—a feature which distinguishes this trouble from one of an inflammatory nature. Passive movements are often impracticable on account of the muscular tension, and the trouble may thus be mistaken for anchylosis. The diagnosis may be established while the patient is under the influence of chloroform, when the free mobility of the joint becomes evident. Esmarch remarks that a creaking noise may often be noticed in the joint.

The foregoing account will sufficiently justify the retention of this affection amongst neuralgic diseases, since it presents all the symptoms by which they are characterized. In fact, almost all authors from Brodie downwards, and especially Esmarch and O. Berger, hold this view. Volkmann alone is somewhat more reserved in his opinion regarding this point.

Articular neuralgia may occur in any joint, though it is far more frequent in the knee and in the hip than elsewhere, and according to the estimates of Esmarch about twice as frequently in the knee as in the hip. The wrist, ankle, and shoulder are, however, not rarely attacked, and even occasionally the small joints of the finger and of the metacarpus. The pain in these cases is generally violent and runs along the digital nerves. As a rule, one joint only is affected, rarely two or more, though this has been occasionally observed.

COURSE.

The course of the disease is usually very uncertain, and in hysterical patients often defies all estimates regarding it. Great variations occur, which may be occasioned by the most diverse external influences. Thus, for example, complete and sudden recovery often follows some powerful mental excitation, as a fall from a horse, attendance upon a ball or wedding, the occurrence of pain in some other part of the body, or the supervention of another disease. The course may, however, also be very obstinate and protracted, the patient being confined to her bed for years, a torment to herself and to all around her; contractions and paralysis also sometimes occur, and the patient may become subject to premature marasmus. When the nervous system is otherwise healthy, the course is usually simpler than in ordinary neuralgia, and after it has lasted for a variable period, recovery gradually takes place.

DIAGNOSIS.

The diagnosis is often beset with no small difficulties because it is frequently quite impossible to determine whether we are dealing with a case of neuralgia or with the earliest stages of inflammation or caries. Esmarch admits that he has often regarded commencing caries sicca of the joint for some time as articular neuralgia. This is, of course, a matter of immense importance in its bearings upon the question of treatment. The differentiation between the two is rendered more difficult when the neuralgic symptoms have developed from unmistakable inflammatory states.

If we are dealing with a well-marked hysterical patient, in whom all the indications of a high degree of nervous excitability are present, the diagnosis is materially facilitated. But if this be not the case, and indeed in all instances, the diagnosis will rest upon the *remarkable disproportion between the* *violence and duration of the affection and the small amount of local disease* (Esmarch). In every case of this kind the examination must be very careful and precise, and we can only decide that the case is one of *neuralgia*, when the pain occurs in paroxysms, when it is for the most part absent at night, when it can be made to disappear by diverting the attention of the patient, when it is not increased by pressing the articular surfaces against each other, when painful points and cutaneous hyperæsthesia are present, when there are vaso-motor phenomena, and finally when rest, abstraction of blood, fixation of the joint rather do harm than good. On the other hand, we may diagnosticate an *articular inflammation*, when there is distinct and persistent swelling, when pain is experienced on pressing the ends of the bones together, when the joint is kept partially bent, when there are febrile symptoms, when cold, abstraction of blood, the application of a plaster of Paris bandage, etc., produce decided benefit. In many cases chloroform narcosis proves very useful in establishing the diagnosis, and, in addition, sometimes causes the pain in neuralgia to disappear for some time; the same is true also of subcutaneous injections of morphia.

Prognosis.

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In forming a prognosis, not the local disorder alone, but the general condition and method of life of the patient must be considered. If she is young and robust, and not of a nervous temperament, the prognosis is favorable; on the contrary, if she is highly nervous and hysterical, the prognosis is doubtful or relatively worse in proportion as the conditions occasioning or leading to the disease are difficult to remove, in proportion as the obstacles to placing the patients under favorable conditions are great, and in proportion as the intelligence and mental energy required to carry out an appropriate plan of treatment are small. These general observations must serve to indicate the points by which the prognosis must be guided.

Treatment.

After what has been said in regard to the nature and pathogenesis of the disease, it stands to reason that in most cases the treatment must be chiefly directed to the general health of the patient; permanent recovery can only be expected after the removal of the cause of the disease. The treatment should at first be of a general tonic character, and should be directed to the removal of the hysteria and allied nervous conditions. Hence iron and quinine, change of air and scene, a residence in some mountainous region, a cold-water cure, ferruginous and brine baths, and especially sea bathing, prove extremely useful; hence, too, diversions of the mind, pleasant occupations, traveling, and removal from home influences, which may be of an irritating and depressing nature, in many instances act favorably. Tact and discretion are required on the part of the medical attendant to fulfil these indications, but such measures, judiciously insisted on, will sometimes promptly effect improvement or recovery. In proof of this the instructive cases of Brodie, Esmarch, and Stromeyer may be referred to. It need scarcely be added that any local disorder tending to produce or maintain the affection should be carefully attended to, especially diseases of the generative organs, dyspepsia, constipation, hepatic disorders, etc.

In regard to the *direct treatment* all authors agree that the ordinary antiphlogistic measures, bleeding, cold, purgatives, blisters, especially when made to suppurate, and issues, are rather harmful than beneficial. The maintenance of the joint in a condition of absolute rest is also useless, and hence it is unnecessary to apply plaster-of-Paris bandages. On the other hand, treatment directed to the mind is essential; indeed energetic psychical treatment often proves instantaneously effective, affording an explanation of the recoveries that take place through sympathy or mesmerism, through the agency of miracle-mongers, relics, the water of Lourdes, etc. In order that this result may be obtained, but two things are necessary, viz., strong faith and a little hocus-pocus. Esmarch justly observes that it is allowable for the physician, under these circumstances, to resort to this kind of influence over the mind of his patient—influence of mental conditions, without at the same time employing any deception. Above all it is requisite that the confidence of the patient should be gained (by careful examination and a confident manner), and then the physician should state boldly that the disease is insignificant, and that it is only necessary that the joint should be used. To convince the patient that this is the case, he should proceed to carry out passive motions, or, better still, to make the patient walk a few steps whilst engaging her in lively conversation, or asking her questions that will make her forget her complaint.

The first steps having thus been taken, if a little good temper be displayed, and energy on the part of the patient, recovery as a rule quickly follows. Strong psychical impressions, which are accidentally brought to bear upon the patient, may in a similar way effect a cure (see cases reported by Esmarch and Stromeyer). These impressions should, however, be followed up and assisted by appropriate local treatment, which, according to Esmarch, should, as far as possible, be of a negative character, since everything that draws the attention of the patient to the affected part makes it worse. The most important thing of all is *exercise of the joint;* in fact, recovery often commences immediately after the first efforts have been made to use the joint again. The movements may at first be passive, and should be quickly succeeded by active ones, even if they cause a little pain. In some cases good results have followed energetic rubbing, which involves a certain amount of passive movement. Cold douches and frictions to the joint usually act favorably, whilst the prolonged influence of cold is injurious. Esmarch speaks very highly of short, cold sea baths continued into the latter part of autumn. Wernher strongly recommends permanent extension in nervous coxalgia; he hangs weights of five or six pounds to the ankles, for the most part without counter-extension, allowing, however, the foot of the bed to be a little higher than the head. The effects were, in many cases, almost magical.

If the patient be otherwise healthy, the ordinary treatment of neuralgia may be adopted. Subcutaneous injections of morphia or atropine are often very efficacious, and flying blisters are sometimes useful. Good results are obtained from the application of electricity (either the faradic brush, or strong galvanic or faradic currents passed through the joint transversely, the course of the main nerve trunks being kept in mind). Amongst internal remedies, the use of which may not be called for by the causal indications, quinine and arsenic are the most important. The treatment of certain symptomatic indications (muscular atrophy secondary paralyses, etc.) requires no special comment.

Anæsthesia.

Treatises of *Romberg*, *Hasse*, and *A. Eulenburg.*—The different manuals of physiology, especially E. H. Weber's article in Wagner's Handwört. d. Phys., 1849, on "Tastsinn und Gemeingefühl."—*Puchelt*, üb. partielle Empfindungslähmung, Heidelb. med. Annal. X. 1845.—*Meissner*, Beitr. z. Anat. u. Physiol. d. Haut, 1853.—*Eigenbrodt*, üb. d. Diagnose der part. Empfindungslähmung. Virch. Arch. XXIII. 1862.—*Smoler*, cutane Analgesie u. ihr. symptomat. Vorkommen in verschied. Krankheiten. Prag. Vierteljahrschr. 1865. Bd. III. u. IV.—*Goltz*, Neues Verfahren, die Schärfe des Drucksinns in der Haut zu prüfen. Centralbl. 1863. No. 18.—*Mosler*, d. Sieveking'she Aesthesiometer. Arch. d. Heilk. 1864.—*E. Leyden*, Unters. üb. d. Sensibilität im. gesund. u. Krank. Zustande. Virch. Arch. Bd. 31. 1864.—*A. Eulenburg*, Thermäthesiometer. Berl. klin. Woch. 1866.—No. 46.—Verfahren zur Drucksinnmessung. ibid. 1869. No. 44.—*Nothnagel*, z. Physiol. u. Pathol. des Temperatursinns. Arch. f. klin. Med. II. 1867.—Z. Lehre von d. vasomotor. Neurosen. ibid. 1867.—*Salomon*, üb. Centrallähmungen. Deutsch. Klin. 1863. No. 36.—*Landois* u. *Mosler*, neuropathol. Studien. (Partial paralysis of sensation), Berl. klin. Woch. 1868.—*O. Berger*, Drei Fälle von partieller Empfindungslähmung. Wien. med. Woch. 1872. No. 27 bis 32.—*Schüppel*, ein Fall von allgem. Anäthesie. Arch. d. Heilk. XV. 1873.

Anæsthesia of the Trigeminus : Kocher, *Hirschberg*, *Guttmann* (Cases of paralysis of the trigeminus); Berl. klin. Woch. 1868, Nos. 10 and 11 ; 48 and 49 ; 51.—*Dixon*, Med. chir. Transact. 2. Ser. Vol. X.—*Snellen*, Arch. f. d. Holländ. Beitr. z. Natur- u. Heilk. I. 1858.—*Büttner*, Ernährungsstörung nach Durchschneidung des Trigeminus etc. Zeitschr. f. rat. Med. 3. Reihe. XV. 1862.—*Hippel*, Ernährungsstörungen des Auges bei Anästh d. Trigem. Arch. f. Ophthalm. XIII. 1, p. 49.—*Meissner*, Zeitschr. f. rat. Med. 3. Reihe. XXIX. 1867.

By the term *anæsthesia*, or sensory paralysis, the diminution or complete suppression of the conduction of stimuli to the mind through the centripetal (sensory) nerves, is implied. If the term be taken in its broadest sense, it includes, first, paralysis of the sensory nerves of the skin (*cutaneous anæsthesia*); next, paralysis of the sensory muscular nerves proper, and of all sensory nerve fibres which belong to the apparatus of motion (*muscular anæsthesia*); sensory paralysis of the nerves supplying the viscera (*visceral anæsthesia*); and, lastly, paralysis of the nerves of special sense (*sensual anæsthesia*). There is considerable difference in the practical importance of these several forms; by far the most frequent and prominent being the paralyses of the sensory cutaneous nerves, which will therefore here be first considered.

Cutaneous Anæsthesiæ.

Cutaneous anæsthesia is extremely common, occurring both as an independent affection, and also,—and this is the more common occurrence,—as a symptom in various diseases of the nervous system. In most instances there is only a *diminution of sensation* (sensory paresis) in the skin, but occasionally the cutaneous sensibility is entirely abolished (sensory paralysis). At the point where the skin no longer presents its normal sensitiveness to ordinary sensations the patient often experiences peculiar morbid feelings, which he is in general unable to describe with accuracy, but speaks of them as sensations of "numbness," "furriness," "crawling," "formication," etc. These are all either due to the blunting and diminution of the normal sensations,—and in that case are only observed when, under the operation of external stimuli, such sensations should normally be felt—or to pathological conditions of recent origin. The latter represent true *paræsthesiæ.*

It is well known that very different kinds of sensations are communicated through the skin, and these are generally divided into two great groups—*tactile sensations* and *common sensations.* In each of these groups manifold varieties of sensation are again distinguishable. To the first group we may refer *sensations of pressure* and *sensations of temperature*, amongst which are included the sensations caused by simple contact of external bodies with the skin (pressure-minima), the various qualitatively distinct sensations caused by the contact of various kinds of bodies, those well-defined sensations of pressure which are susceptible of measurement by the mind, and, lastly, those produced by differences of temperature.[1]

In the second group,—that of common sensations,—very different kinds of sensation are included; the best known of these are pain, itching, titillation, sensual pleasure, and that resulting from electrical excitation.

The skin, however, does not only communicate all these varieties of sensations, but we receive at the same time information regarding the place where the outward irritation has been applied to the skin, and by virtue of the sense of locality, we are able to recognize several coincidently applied and qualitatively similar sensory excitations as distinct.

The relations of the cutaneous sensibility thus become both complicated and difficult to understand, and in pathological cases the different sensory qualities, such as the tactile feeling, and the feeling of common sensation, the different senses of pressure, of temperature, and of locality, as well as the ordinary feelings of pain and itching, and the electrical sensation, must all be closely investigated.

The sensory qualities are not always paralyzed at the same time, and in the same degree, in pathological conditions. But if this be the case and if every kind of sensation be lost, the case is said to be one of *total sensory paralysis*. In many instances, however, only particular kinds of sensation are diminished or abolished, the others remaining more or less perfectly preserved;

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[1] There might perhaps be some practical advantage in distinguishing those kinds of sensations that result from the gentle contact of external bodies (stroking, touching with smooth, rough, dry, damp, woollen, and similar bodies), and those caused by the application of variously shaped bodies (for instance, pieces of money, rings, keys, etc., the form and quality of which are easily recognized without any estimate being made of the pressure exerted), as *tactile sensations proper* (Tastgefühle). or as *sensations of contact* (Meissner's simple tactile sensation), from the genuine *sensations of pressure* which indicate the amount of and differences in the pressure exerted. It cannot at the same time be denied that, strictly speaking, all these tactile sensations are referable to modifications of pressure and temperature (aided by the sense of locality). Our only intention is to point out that for practical purposes, and particularly for the purposes of diagnosis, some advantage would be gained by making the above distinction.

for instance, tactile sensations may be extinguished, and the ordinary feelings be preserved, or *vice versa*, and such a case would be termed one of *partial sensory paralysis.* Moreover, particular tactile sensations or particular feelings of common sensation may be lost or preserved, and then *partial tactile paralysis* and *partial paralysis of common sensation* are said to exist. As might be expected, all possible combinations may present themselves, and have indeed, for the most part, been already observed. The sense of pressure, again, may be alone lost, whilst the remaining sensations are preserved, and the same may occur with the senses of temperature and of locality, or two of these sensations may be paralyzed, whilst the third is unaffected; and with all these there may or may not be simultaneous paralysis of ordinary sensation. Amongst the partial paralyses of common sensation, that which most frequently occurs is loss of the sensation of pain (*analgesia*), which may occur either with or without partial paralysis of the tactile sense. It is thus obvious that the number of possible combinations is very considerable, and that it is only by careful investigation that the kind of paralysis present can be accurately determined. In many instances the several combinations merge in one another, and, with the progress of the pathological changes, the sensory paralysis may be observed to extend itself over a larger and still larger area, and in like manner partial sensory paralysis may, by passing through a series of intermediate degrees, gradually become total. In regard to the extension of anæsthesia over the surface of the skin, a distinction is made between *circumscribed* (local) and *diffused*, or general, anæsthesia; we speak too of unilateral and bilateral hemiplegic and paraplegic sensory paralyses, etc., expressions that require no further explanation.

In regard to the intensity or degree of disturbance, the term *complete* anæsthesia is applied to that condition in which some particular sensation is entirely extinct; *incomplete*, in which it is only more or less diminished.

Pathogenesis and Etiology.

In order to comprehend the pathogenesis of anæsthesia, it will be expedient to consider briefly the physiological processes constituting the basis of sensation.

All sensations derived from the skin originate in a change of the peripheral terminal apparatus of the sensory cutaneous nerves, with the structure of which we are as yet only partially acquainted (Vater's or Pacinian corpuscles; Meissner's tactile corpuscles, the club-shaped terminal enlargements of Langerhans). Whether these several forms of terminal apparatus are adapted to receive and communicate the different kinds of sensations, is still unknown.

Every external influence acting upon the terminal apparatus constitutes a stimulus, provoking irritation; and this irritation is transmitted through the sensory nerve-fibres, in a centripetal direction. Here again the question still remains undecided whether there are different paths of conduction for the several sensations. Neither the reasons adduced by many physiologists, nor the pathological cases reported by numerous observers (Nothnagel, Berger), which appear to favor the view that such different paths do exist, permit any positive statement to be made on the point at issue. It is even somewhat doubtful whether conduction to the central organs from the peripheric terminal apparatus takes place in a perfectly isolated manner. Pathological cases, and the physiological experiments of Arloing and Tripier have rendered it probable, in regard to the terminal ramifications of the sensory nerves of the upper extremities, that after the destruction of a particular sensory nerve, the sensory impressions formerly transmitted through it may be conducted through adjoining nerves to the central organ, so that no anæsthesia need necessarily be present in the region supplied by a divided sensory nerve, and we are driven to admit the existence of numerous peripheric anastomoses between the several sensory nerves.

The peripheric paths of conduction enter the spinal cord by way of the posterior roots. But some obscurity exists in regard to their subsequent course in the cord. The posterior white columns (and perhaps also the adjoining parts of the lateral columns) and the gray substance must, in any case, be the principal means of conduction. But the relation of these parts to the several sensations has not as yet been clearly ascertained, nor is it known whether separate nerve paths exist for them or not. Schiff was the first to discover that the presence of the gray substance is requisite for the conduction of the sense of pain, and that the posterior columns, even when isolated, are still capable of transmitting tactile sensations. Schiff was consequently led to infer the existence of distinct conducting paths for these sensations. A view that has lately been advanced is to the effect that the principal conducting paths run in the white substance and in its immediate neighborhood, whilst in the gray substance there are only secondary conducting paths. These last, according to this view, are used for conduction only when powerful stimuli are applied, but then they cause strong excitation (pain). The greater number of the sensory conducting paths decussate in the spinal cord, though at different heights.

In the medulla oblongata the sensory paths are more laterally situated; they run through the pons and the crura cerebri, then enter into relations that have not as yet been satisfactorily ascertained, with the great central ganglia, and especially with the corpora quadrigemina, and finally, forming part of the corona radiata, extend to the cortex cerebri. The sensory fibres appear to terminate chiefly in the cortical region lying behind the Sylvian fissure, and it is here, therefore, that we must look for the central terminal apparatus for the reception of sensory impressions. Whether there are different paths of conduction as well as different central organs in the brain for the several kinds of sensory qualities, has not as yet been clearly ascertained, but certain pathological observations made by Mosler, Berger, and others have at least rendered it probable.

A glance at the above shortly-sketched relations shows at once that anæsthesia may arise from changes in very different parts of the sensory nervous apparatus: as 1, from some change (inexcitability) in the peripheral terminal apparatus; 2, from inhibition or interruption of the conduction of sensory impressions, which may take place at some point, *a*, of the periphery, or *b*, of the spinal cord, or *c*, of the brain; lastly, 3, from some change (inexcitability) of the central organs of reception. In accordance with these possible variations, the *causes* of anæsthesia may be grouped in a very simple manner into:

1. *Causes which lower or destroy the excitability of the peripheral sensory terminal organs* (true peripheral anæsthesiæ).

One of the most palpable causes, and the anæsthetic effect of which is most frequently brought under our notice, is undoubtedly *cold* (local deprivation of warmth). As a typical instance of its action, the local anæsthesia, caused by the application of ether spray, or some similar agent, to the skin, may be mentioned. In consequence of the intense cold thus produced, the skin becomes in a short time so insensible, that surgical operations can be performed on it without pain. This method of preventing pain, as is well known, is in favor with many practitioners for the smaller operations. The application of ice or of cold mixtures, as well as long exposure to very cold air, produces the same effects.

In this way the excitability of the peripheral terminal apparatus, and perhaps also of the sensory nerves immediately adjoining, becomes lessened or entirely abolished. The physiological experiments of Weber, A. Eulenburg, Nothnagel, and others, have demonstrated that the sensations of pressure, temperature, and space, the feeling of pain, and the sensibility of the skin to electric stimuli, may all be lessened in a similar manner. Complete congelation causes loss of sensibility, by the formation of an eschar; the so-called "rheumatic anæsthesiæ" are traceable to other causes rather than to the direct effect of cold. In like manner the influence of *high degrees of heat* may lead to a blunting diminution of the perception of the different kinds of sensation as has been demonstrated by the experiments of Weber and Nothnagel.

It may frequently be observed that the protracted action of certain corrosives on the skin diminishes its sensibility: the best known example of this kind of anæsthesia is that which occurs in laundresses (Romberg) from the action of alkaline salts, though the temperature of the water has also much to do with its origin. A similar effect has been noticed as a result of the action of powerful acids (acetic acid, aqua fortis, &c.); no doubt can exist that this is due to the action of these corrosives upon the peripheral terminal apparatus, even though we may be unable to explain the particular mode in which they act.

Electrical currents are also capable of lessening the sensibility of the skin to some extent. Nadedja Suslowa found that a diminution of the tactile sense, as well as of the perception of space and temperature, was observable after the transmission of a weak galvanic current through the skin and in the area of the skin adjoining the anode of the galvanic current; but he observed no change in the acuteness of common sensation.

The experiments that were made by A. Eulenburg with the subcutaneous injection of morphia, show that *certain narcotics* directly lessen the sensibility of the skin. The energetic application of ointments containing atropine, aconitia, &c., may also diminish the sensibility of the skin, apparently by their influence on the peripheral terminal apparatus.

Disturbances of the circulation are of equal importance in producing anæsthesia. *Ischæmia* of the skin is a particularly frequent and effective cause of cutaneous anæsthesia. Both the mechanical and the spasmodic ischæmia are capable of lowering the excitability of the peripheral terminal apparatus, and many vaso-motor neuroses (spasmodic contraction of the arteries); which are accompanied by anæsthesia (Nothnagel), act in this way. The acuteness of the tactile sense may also be lowered by *passive congestion* (Stauungshyperæmie) (Alsberg). The mode in which the local anæsthesiæ observed in many affections of the skin (as for example in the multiform circumscribed cutaneous paralysis described by Veiel, and in Herpes Zoster) arise, has not been clearly ascertained; though in the case of Zoster, we may, with some probability, ascribe it to arrest of conduction in the peripheral nerves from neuritis. Lastly, *complete destruction of the skin* (owing to burns, frost-bites, ulceration, &c.), by destroying the peripheral tactile organs, abolishes also those kinds of sensations which they are destined to transmit.

2. *Causes arresting or inhibiting conduction in* PERIPHERAL NERVES. (First group of anæsthesiæ of the conducting organs). *Cold* may here also constitute a cause; its influence on peripheral nerve trunks being to lower the acuteness of sensation through-

out the area of their distribution. Cold, however, generally produces anæsthesia by its action on the peripheral nerve trunks: in most cases of this kind it is probable that a slight ("rheumatic") neuritis is present. In the next place, *injuries* (of all kinds and in the widest sense of the term) are frequently causes of peripheral anæsthesia; but an enumeration of the possible varieties would lead us too far. In many instances simple compression has only been effected, as in the case of herniæ, tumors of all kinds, neuromata, lepra nodes (in lepra anæsthetica), etc. Simple neuritis, if the nerve fibres undergo compression to a sufficient extent, may lead to anæsthesia, as occurs in Zoster; in a case related by Remak, after a wound of the nerves of the ungual phalanx of the thumb, a centripetal anæsthesia of the parts supplied by the radial and median nerves gradually supervened,—a result that is explicable on the supposition that it was a case of progressive neuritis; the suggestion made by Remak that it was due to reflex anæsthesia is, in our opinion, much less probable. Some of those anæsthesiæ that accompany neuralgiæ belong to this category, while others are best explained by admitting the existence of some influence acting on the central organ (see above).

3. *Causes which occasion arrest or suppression of conduction in the* SPINAL CORD. (Second group of anæsthesiæ of the conducting organs.)

It is very difficult to refer spinal anæsthesiæ to any particular local change in the spinal cord, first, on account of the unsatisfactory state of our knowledge in regard to the physiological conducting paths; and, secondly, on account of the imperfect information we possess in regard to the pathology of the cord itself. The chief seat of partial sensory paralysis is undoubtedly the central organ; for the majority of cases belonging to this category depend upon disease of the central nervous system, whilst there are comparatively very few clear and indubitable instances of partial sensory paralysis due to disease of the peripheral nerve trunks on record, if we except the case observed by Nothnagel (loc. cit.), which, however, was first examined two years after the injury was inflicted. For all details on these points we must refer to the description of the diseases of the spinal cord, which will be found in a subsequent volume of this Cyclopædia, and a few remarks must here suffice.

The effects of complete division or compression of the spinal cord are, of course, very simple, consisting of total loss of sensation of all those parts of the skin which receive their nerves from the cord below the situation of the lesion. This condition is observed in fractures of the vertebral column, in cases of destruction of the spinal marrow from extravasation of blood, from tumors, kyphoses (or curvatures), and congestion-abscesses, and after division or laceration of the cord, etc. Partial sensory paralyses must be referred, on the contrary, to diseases of the posterior (and in part of the lateral) columns, or of the gray substance, or of both together. As appears from Schiff's most recent observations,[1] not only the extent of the disease, as seen in the transverse section of the spinal cord, but its position at various planes of its height, is of importance in regard to the amount and kind of the sensory disturbances produced, since the fasciculi, by which conduction of sensory impressions is effected, gradually alter their position (the fibres which run in the lateral cords, for example, passing from them into the posterior cords of the gray substance), in consequence of which the relations become still more complicated. It, however, appears to have been clearly ascertained, that in disease of the gray matter, common sensation, in disease of the posterior columns, and of the lumbar portion of the lateral columns, tactile sensations, are chiefly impaired; moreover, that in uni-lateral spinal anæsthesia, the existence of some lesion of the opposite half of the spinal cord may be assumed (in consequence of the decussation of the sensory fibres in the spinal cord). To what precise anatomical seat the partial paralyses of tactile sensibility, so frequently observed in diseases of the spinal cord, are to be referred, we are still entirely ignorant.

From this point of view it is easy to understand that disturbances of the sensibility are amongst the commonest symptoms of the different diseases of the cord, and that the most manifold degrees and combinations of such sensory disturbances are observed which are conditioned by the seat and extent of the spinal affection, and that in point of fact all possible forms of partial sensory paralysis occur. Tabes dorsalis, chronic myelitis, sclerosis, and softening of the spinal marrow are the affections that are most frequently associated with such anæsthesiæ; and for all further particulars, we must refer to the detailed description of these diseases in a subsequent volume.

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[1] Centralblatt für die Med. Wiss., 1872, No. 49.

4. *Causes which occasion arrest or interruption of the conduction in the* Brain. (Third group of anæsthesiæ of the organs of conduction.)

The conditions are here just as obscure and as difficult to understand as in the case of the spinal cord. The existence of separate conducting paths for the different kinds of sensations appears to be deducible from numerous pathological observations; no certain facts have, however, been obtained in regard to the position or course of these channels of conduction in the brain, and it is probable that we shall only gradually obtain accurate information on this point by numerous and exact observations made on cases that happen to occur. At present it can only be said that the sensory nerves may be injured in any part of their course from the medulla oblongata, through the crura cerebri, the central ganglia, and the corona radiata, to the cortex cerebri, and that such lesions, which may be very various in kind, may lead to anæsthesia. As a result of the complete decussation of all sensory fibres (which has already taken place in the cord), the anæsthesia always occurs on the side of the body opposite to the cerebral lesion. It generally assumes an hemiplegic form, though, of course, with bilateral disease, bilateral anæsthesia also occurs; and, on the other hand, if the cerebral lesion be small, the anæsthesia may be quite limited. Motor paralysis (Hemiplegia), and various symptoms indicative of disease of the brain, are almost always present. For all details respecting these points, the reader is referred to a subsequent volume of this work.

We shall here only add, that hæmorrhage into the cerebral substance constitutes the most frequent cause of anæsthesia of the conducting fibres of the brain; and this is especially the case when the hæmorrhage takes place into the pons, the crus cerebri, the external parts of the optic thalamus, and the adjoining fibres of the corona radiata, though the anæsthesia is in most cases of relatively short duration. Well-marked anæmia of the brain (such, for instance, as may be produced by embolism), softening of the cerebral substance, encephalitis, sclerosis, tumors, wounds, &c., and, in short, all conditions, capable of disturbing or interrupting the conduction through sensory fibres, act in the same manner.

5. *Causes which diminish or destroy the excitability of the central receptive apparatus.* (*True central anæsthesiæ.*)

These include lesions of those parts of the brain which effect the translation of sensation into consciousness, and probably, therefore, lesions of the gray cortex of the brain, and particularly of its posterior portions. Every kind of anatomical lesion of these parts may occasion anæsthesia. Diseases of the meninges are particularly liable to produce anæsthesia, by causing disease of the gray layers of the convolutions, and an explanation is thus afforded of the exceedingly frequent occurrence of anæsthesia in many forms of mental disease, the seat of which we are disposed to locate in the cortex itself.

Amongst cerebral lesions it is particularly chronic encephalitis, with its different terminations in sclerosis, or in softening, embolism, meningeal apoplexy, inflammations of the meninges, etc., that is apt to be associated with central anæsthesia. The anæsthesia that is associated with epileptic attacks, catalepsy, sopor, uræmia, coma, etc., may also be attributed to disturbances of the functions of the cortical portion of the brain. Amongst the true psychoses, anæsthesia (and also, in particular, analgesia) appears most frequently in melancholia attonita and paralytic idiocy,—in the latter case, however, being in part also due to the coincident disease of the spinal cord. Anæsthesia is also not uncommon in simple melancholia, in insanity, and in the different forms of idiocy. We are thus led to understand why patients suffering from mental disease often bear the most painful operation with the greatest equanimity, and even mutilate themselves in the most horrible manner. For details, the special works on this subject must be consulted.

But there are still numerous forms of anæsthesia, in regard to the pathogenesis and seat of which we possess but little information, and which, therefore, have not been considered under any of the foregoing heads. The most prominent of these is the anæsthesia that occurs in connection with hysteria, of which it forms one of the commonest symptoms. It sometimes appears to be quite local, whilst at other times it extends over one-half or even over the whole surface of the skin, either as a complete, or, less frequently, as a partial paralysis of sensation, and particularly frequently as an analgesia. We are still ignorant of the true seat of hysterical anæsthesia; it is probable that, in the majority of cases, the central nervous system is affected, though the particular part may vary considerably. The seat of the form of anæsthesia which is sometimes observed as an accompaniment of epilepsy, catalepsy, chorea, hypochondria, and other neuroses, is also not clearly ascertained.

The so-called *toxic anæsthesiæ* are generally due to some lesion of the central nervous system; at least, this may be said of the sensory paralyses produced by the so-called *anæsthetics* (chloroform, ether, liquor hollandicus, etc.) It appears fairly certain that these agents first lower the excitability of the sensory centres, and then that of the motor and reflex centres; but probably the peripheral nerves and terminal apparatus do not remain unaffected. The anæsthetics act particularly by effecting a change in the gray substance, whilst the nerve fibres appear to be less readily influenced by them. This, perhaps, affords an explanation of the fact that, in a certain stage of the narcosis, tactile sensations can still be perceived whilst the sensation of pain is already abolished.

The anæsthetic action of *narcotics*, such as morphia, atropine, etc., is much less marked; still, however, even when administered in small doses, they blunt the sensibility, and, in large doses, produce sopor and complete anæsthesia. Their mode of action is almost identical with that of the anæsthetics. The action of different toxic substances, such as alcohol, lead, copper, arsenic, ergotine, saponine, bromide of potassium, and others, in producing anæsthesia, has been hitherto a pure matter of supposition, and a wide field still remains for observation and investigation.

After acute diseases anæsthesiæ often appear, sometimes as local, sometimes as more or less widely distributed affections, in regard to the localization of which opinions are not as yet settled. Such conditions usually occur after typhoid fever and diphtheria, but have also been observed after other infectious diseases, such as cholera, dysentery, scarlatina, etc., and even after simple inflammation, accompanied by febrile symptoms. The seat of the disease probably varies in different instances, the central as well as the peripheral portions of the nervous system being affected. Motor paralyses are usually associated with the anæsthesia.

Lastly, highly important statements have very lately been made by Fournier,[1] in regard to the unusually frequent occurrence of analgesia with syphilis in its secondary stages. In women this condition is a very common symptom of secondary syphilitic affections, and whilst it may be generally diffused over the whole body, it is more frequently quite local, appearing in the form of islands in different parts of the skin. The most favorite places are the backs of the hands and the breasts. It is seldom associated with obtuseness of the tactile sensibility, and still more rarely with a diminution of the sense of temperature. Its dependence on syphilitic infection is strongly asserted by Fournier, who clearly indicates the points that distinguish it from hysterical analgesia. We are still completely ignorant of its anatomical seat.

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Symptoms.

Anæsthesia discloses itself by subjective as well as by objective symptoms ; the former are, however, frequently very indefinite and ambiguous, and for a knowledge of the exact nature, extent, and seat of the disease, the objective symptoms are by far the most valuable. These must be ascertained by careful objective investigation. Unfortunately this is very difficult and troublesome, occupying much time, and being also, in many instances, and especially with uneducated patients, very unsatisfactory. In many cases, of course, the results are sufficiently obvious and intelligible, but if we reflect on the paucity of our knowledge in regard to the most important points of the physiology and pathology of the sensory apparatus, in spite of the manifold investigations that have been undertaken, we must acknowledge that much valuable time and labor have been already uselessly expended over these observations.

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[1] Leçons sur La Syphilis. Paris, 1873, p. 783, *et seq.*

A short account of the methods of testing the sensibility will not here be out of place. As a preliminary, the fact first discovered by Türck may be mentioned, viz., that the district affected by anæsthesia appears larger when the investigation commences from the anæsthetic district of the skin, and advances towards the healthy part, and smaller when the examination is undertaken in the opposite direction. The limits, therefore, between the healthy and diseased districts alter according to the direction in which the investigation is made. This may be observed in each case.

The investigation of the true *tactile sensations* or *feelings of contact* (see remarks on p. 200), may be most readily accomplished by delicate and superficial contact of foreign bodies, as by moving the tips of the fingers or the head of a needle over the skin, and by touching the hairs. Thus, for example, it may be ascertained whether the patient can distinguish the head from the point of the needle or whether he can determine the nature of smooth, rough or woollen objects. In those parts which are more especially used for the determination of the finer distinctions between different objects, as, for instance, the palms of the hands, the test may be made by placing various bodies (pieces of money, small keys, buttons, rings, etc.), upon them, and making the patient describe them with closed eyes. By a parallel series of experiments on healthy persons (and, best, on one's self), a judgment may be acquired as to what the discriminating power of the tactile sense is in a given region, thus enabling us to draw correct conclusions in pathological cases. Where the disease is unilateral, the easiest and most certain method is to compare the two sides together.

The testing of the *sense of pressure* is best effected by the superimposition of weights, in order to determine the minimum differences that can be perceived (E. H. Weber's method). This method is so well known that it is unnecessary to enter into further details in regard to it. The testing can usually be carried out with facility by means of coins (Eigenbrodt), such as silver crowns or half dollars, a larger or smaller number of which can be placed in the patient's hand. Certain precautions, however, require to be taken, namely, the exclusion of the muscular sense, which may be effected by resting the part of the body to be investigated upon a firm support; the exclusion of the sensation of temperature by intercalation of a bad conductor of heat, as, for instance, small wooden disks; and lastly, the weights should be placed on the part at regular intervals of time.

Eulenburg has materially facilitated the testing of the sense of pressure by constructing a baræsthesiometer (measurer of the sense of pressure), on which, by varying pressure upon a spring, different degrees of pressure may be read off on a dial. The instrument is handy, and can be applied to any part of the skin, though here, also, the muscular sense must be excluded. Eulenburg found, by means of this instrument, that a difference of pressure, amounting to from $\frac{1}{30}$ to $\frac{1}{40}$, can be discriminated by the skin of the face; on the hand and arm it amounted to from $\frac{1}{20}$ to $\frac{1}{10}$; on the anterior aspect of the thigh and leg, and on the dorsum of the foot, the discriminating power is about the same as on the hand; finally the perception of differences is much less acute on the skin of the sole of the foot and on that of the back of the leg. These numbers may serve as a standard by which to judge pathological cases. Quick and certain results are only obtained in unilateral affections, in which the two sides of the body can be compared; in other instances, the greatest care and precision are requisite to demonstrate the existence of slight anomalies of the sense of pressure. The method of testing the sense of pressure suggested by Goltz, by means of waves of various strengths generated in a tube of gutta percha filled with fluid, is scarcely likely to be introduced into practice.

The sense of temperature may be roughly tested by the simple application of hot and cold bodies (hot or cold metal rods, test tubes filled with hot and cold water), but a more delicate means of testing it is to see whether the patient can distinguish with certainty the difference between cool currents of air, such as may be produced by blowing on the part from a little distance, and warm ones, such as may be produced by breathing upon it in close proximity. For a still more delicate investigation, instruments must be used which are capable of indicating minute differences of temperature. Nothnagel's instrument consists of two cylinders, with metal bottoms filled with water heated to different degrees; Eulenburg's instrument consists of two thermometers provided with large, flat, quicksilver bulbs, suitable for placing on the skin, and which are attached to a stand in such a manner that they can be fixed at different heights (thermæsthesiometer). Variations of temperature (between 80° F. and 91° F.), amounting to half a degree or less, are almost always discriminated most accurately. On the cheek, a difference as small as one-third of a degree (Fahrenheit) can be distinguished, and on the back, one of about a degree and five-ninths. Deviations, to any considerable amount, from these numbers, must be regarded as pathological. Alternate immersion of the fingers, hands, or feet in water at different temperatures, may in some measure supply the place of these finer methods of investigation.

Testing of the *sense of locality* (sense of space) is effected in the simplest manner by touching some part of the skin with the finger or the point of a needle, whilst the patient's eyes remain closed, and then directing him to indicate the point touched ; the error in healthy persons is about equal to the minimum distance at which, according to E. H. Weber's method, the points of the compasses can be recognized as separate in different parts of the skin. This method, in which compasses are used, as suggested by Weber (or the analogous æsthesiometer of Sieveking), gives results that are more exact, and admit of being expressed in numbers. By its means we endeavor to discover the smallest distance at which two tactile impressions, separated from each other by a certain space, can be felt as two. This distance varies considerably in different parts, and can be immediately read off on the circle. Any increase beyond the norm of these distances, indicates a diminution of the sense of locality. The nominal distances have been ascertained with great care by Weber, and the following may be here mentioned : the tip of the tongue—1. 2 mm. ; the extremities of the fingers—2. 25 mm. ; the dorsal side of the first phalanx—16 mm. ; the back of the hand—31 mm. ; the forearm and leg—about 40 mm. ; the back—from 40 to 70 mm. ; the upper arm and thigh—77 mm. Of course only considerable deviations from these approximatively normal numbers can be regarded as pathological ; the individual differences being very great, whilst the sense undergoes great temporary improvement by practice. For the details of this somewhat slow method, and for the precautions that should be taken, reference must be made to physiological works.

Amongst the tests of *common sensation*, those for sensation of pain are most important. This can easily be tested by the prick of a pin, by burning, pinching, firm pressure, and also by the application of a strong faradic current. The *sensation of tickling* may be investigated in the ordinary way. A method, that at first sight appears very appropriate, is the test for the *electro-cutaneous sensibility*, by means of the faradic current, as proposed by Leyden. In this it is sought, by means of a pair of electrical tactile compasses, to ascertain the minimum strength of the current required to produce an electrical sensation : at the same time provision is made for reading off the distance separating the coils of the induction apparatus, thus rendering it possible for a comparative numerical estimate to be formed. Unfortunately, this method is very uncertain in its results, chiefly on account of the varying thickness and conducting power of the epidermis, which introduces a very uncertain factor into the experiment. From the results of my own experiments with it, I cannot consider it as being adapted for ordinary use ; and it has the additional disadvantage of occupying a great deal of time. For ordinary cases, a simple application of the ordinary electrodes with the common faradic current is quite sufficient.

In some cases, the investigation of the sensibility of the skin may be rendered more complete by determining *the period required for the conduction of sensations*, and it will be found that cases of retarded conduction of sensation are not very uncommon. If the retardation be considerable, it will be shown by the much longer interval that elapses, as compared with the normal condition ; the patient, in both cases, recording his perception of a simple tactile impression by an exclamation, or by raising the finger. For the determination of still finer differences, the method of physiological research must be adopted.

By all these different methods we can acquire a perfect objective conception of the kind, degree, and extent of the anæsthesia, and we can determine with great precision how far the various kinds of sensation are severally affected. It is easy to understand that the most varied combinations and degrees of disturbance may occur, which it is unnecessary to discuss at greater length. It would be very difficult, indeed, to give a complete picture of the general symptoms of disease in the different forms of anæsthesia, for they differ in each case, and such a picture is to be obtained from clinical observation alone.

The *subjective symptoms* of anæsthesia are, for the most part, either negative, being founded on the diminution of normal sensations, or they are accidental, that is to say, they do not properly belong to anæsthesia, but to accompanying disturbances, induced by the same causes.

Usually the patient complains of a feeling of numbness, "furry-feeling" (Pelzigsein), as if there were something between the skin and the bodies touching it; the fingers seem as if they were covered with gloves, and the feet feel as if the patient walked upon wool, or on a carpet, or on a bladder filled with water, etc. The slighter degrees of this sensation can be most readily perceived in the extremities of the fingers, particularly on rubbing them against each other. In the higher degrees the patient states that he feels as if his limbs were altogether absent, and as if, in walking, he stepped on air; he needs the help and control of the eye. The usual feeling of the contact of dress and of the shoes is lost; in any kind of manual labor the usual sensations are wanting; pens and needles fall out of the hand unless the patient incessantly follows its movements with the eye, and the more delicate occupations become either very difficult or impossible.

Even in complete anæsthesia dull sensations, which the patient is otherwise unable to describe, are sometimes experienced in the deeper-lying tissues and organs, as in the muscles, joints, and bones; these of course are mentioned by him among other subjective symptoms. There is generally a feeling of dull heaviness in the anæsthetic parts. The patient, moreover, very generally complains of itching and formication in the parts affected.

These sensations, however, do not properly belong to anæsthesia, but must be included amongst the *concomitant phenomena* of the disease (since it often happens that they appear independently as an accompaniment to some other affection, as, for instance, neuralgia). The coincident or associated phenomena may vary greatly, and usually depend on the participation of other nerves in the affection, but partly also on the more or less complete abolition of the physiological functions of the nerves implicated.

Thus we find that the *parœsthesiœ* already mentioned (itching, formication, and, more rarely, curious feelings of heat and cold, burning, etc.) are experienced in sensory nerves. These sensations occur, for the most part, though not exclusively, within the anæsthetic areas, though the greatest possible variations occur in this respect. It has not been clearly ascertained what are the conditions on which these sensations really depend; we do not know what kind of irritation is required to excite in the nerves those curious feelings that are described as formication and numbness; we assume, however, that they are essentially caused by certain excitations of the nerves, which, in accordance with the law of excentric projection, are transferred to the periphery like those excentric pains which we shall now proceed to describe. The feelings of heat and cold may perhaps be caused by associated changes in the state of the blood traversing the affected parts.

Amongst the concomitant sensory phenomena, *pain* is a very common symptom. It originates in cases of anæsthesia of the nerve, in consequence of some irritation affecting the portion of the nerve which is situated centrically to the cause of the arrest of conduction, and which, according to the law of excentric projection, is transferred to the periphery, where it may be felt as a very acute pain. The singular circumstance, that parts in which neither pain nor even a sensation of touch can be elicited by any mode of irritation, may become the seat of the most violent pains, has led to the establishment of a distinct form of anæsthesia—the so-called *anæsthesia dolorosa.* No particular advantage, however, is gained by such a subdivision.

It is obvious that excentric pains of this kind can only occur in anæsthesia of nerve trunks, and not in true central anæsthesia, since in the latter case the sensory terminal apparatus is incapable of being excited, and cannot therefore experience any kind of sensation. Indeed, such pains are often observed to occur in all conceivable forms of conductive anæsthesiæ, but they are most common in injuries of the peripheral nerves (as in gunshot wounds, contusions, neuritis, etc.), and it depends more or less upon the casual grouping of the symptoms, whether such cases are to be regarded as neuralgia, anæsthesia, or paralysis; the variety in the symptoms being caused by the associations of the different kinds of nerves in the same nerve trunk.

The *sensations of special sense* are in many instances materially modified, particularly in centric anæsthesia, or in lesions of those nerves which contain nerves of special sense, as in the case of the lingualis. Loss of taste and loss of smell are most common (as in the cases reported by Binz and Guttman); loss of hearing is less frequent, and that of sight very rare. These effects, of course, depend exclusively upon the seat of the disease.

Amongst the *concomitant motor phenomena,* paralysis is one that is very often associated with anæsthesia, and it in general arises from the same causes as those which produce the anæsthesia itself; thus, for example, in mixed peripheral nerves, and in spinal disease, paraplegia, or the disturbance of co-ordination termed ataxia, may be observed, and in cerebral affections, besides sensory, motor hemiplegia may be present. *Cramps* of the muscles are more rarely observed, but may, nevertheless, be occasioned by the primary disease; states of muscular tension, slight fibrillar contractions, spasms, contractures, etc., are for the most part met with in spinal and cerebral anæsthesiæ. Persistent muscular contraction [contracture] may likewise be observed as a result of coincident paralysis in the peripheral forms of anæsthesia.

It is of great importance to study the behavior and relations of the *motor reflex actions.* All reflex actions in peripheric anæsthesiæ are, without exception, abolished, but in spinal anæsthesiæ everything depends upon the seat and extent of the principal disease ; if the arc of reflexion, situated at a lower plane than the disease, remain unaffected, the reflex acts may be preserved and even be increased in their intensity ; if, on the contrary, the arc lie above the part, or be implicated in the disease, the reflex acts are abolished. In cerebral anæsthesiæ the various spinal reflex actions are all preserved, and, indeed, are often performed with abnormal energy, whilst those effected in the cerebrum may undergo modifications in character similar to those which occur in spinal diseases. This is not the place to discuss these points in detail, but they will be considered under the head of diseases of the brain and spinal cord.

In many cases well-marked concomitant *vaso-motor phenomena* are observed : either redness and increased temperature of the skin, sometimes with augmented perspiration, or pallor and coldness, with abnormal dryness of skin. These varieties depend, as in neuralgia, upon the coincident implication of the vaso-motor nerves, which may of course be affected in many different ways in the various forms of the disease. It must be noticed, that anæsthesia *per se*, that is to say, the abolition of the ordinary centripetal excitations, has no influence upon the peripheric circulation, for this can only be altered by direct irritation applied to the vaso-motor nerves, or in a reflex manner by conditions of excitation calling forth in anæsthesia excentric pains and paræsthesiæ.

The same observations apply to *trophic disturbances*, which not unfrequently form concomitant symptoms in anæsthesia. Amongst these are included atrophy of the muscles and other tissues, desquamation of the epidermis, abnormal and irregular growth of the nails, swelling and glossiness of the skin ("glossy fingers"), herpetic eruptions, pemphigus, and ulcers, which have all been observed ; also slight and severe degrees of bed-sores, as well as feeble resistance to all kinds of external injuries, which may be speedily followed by malignant inflammations, ecchymoses or mortification. The simple fact that complete anæsthesia, such as occurs in most cases of cerebral origin, in some of spinal origin, and in hysteria, may run its course without any such trophic disturbances, sufficiently demonstrates that they are not occasioned by the anæsthesia. At most the anæsthesia can only be regarded as a condition likely to lead to trophic disturbances by interfering with the recognition and sufficiently early removal of external injurious agents. These disturbances, however, there can be no doubt, are due to the implication of trophic and vaso-motor nerves in the disease. If the disease be situated where sensory and vaso-motor (and trophic) nerves lie close together, then are the conditions the most favorable for all the nerve fibres to be equally affected ; this explains why such trophic disturbances are most commonly observed in severe lesions of mixed peripheral nerves. In the spinal cord where the sensory and vaso-motor trophic nerves are not in such immediate proximity, they may be separately affected ; which explains why trophic disturbances are so often absent in spinal anæsthesia, whilst they are present in other forms. They are certainly present, however, in all cases in which the entire thickness of the cord is destroyed throughout any considerable extent. On the other hand, in the brain these different nerves lie at so great a distance from each other that it is a rare occurrence for them to be coincidently diseased ; and consequently, as a rule, trophic disturbances do not occur in cerebral anæsthesiæ.

One of the most interesting trophic disturbances, which is not unfrequently observed in trigeminal anæsthesia, is the so-called "*neuro-paralytic ophthalmia*," in the more serious cases of which gradually increasing hyperæmia of the conjunctiva occurs, with cloudiness and ulceration of the cornea, proceeding ultimately to perforation and destruction of the eye by panophthalmitis.

This trophic disturbance, which appears so constantly after the intracranial section of the trigeminus, has been thoroughly investigated by physiologists (Magendie, Büttner, Meissner, Snellen, Schiff, Hippell, and others) ; and it was believed that the ultimate conclusion for or against the existence of trophic nerves would probably be arrived at from observations in this region. The view held by some that the sensory fibres exert a direct trophic influence on the parts they supply has been refuted, and the opinion, so persistently maintained by Snellen, that the loss of the protection ordinarily afforded to the eye by the sensory nerves is the only cause of neuro-paralytic ophthalmia, has also been shown to be untenable. The most probable view is that which was originally advanced by Meissner, viz., that the first branch of the trigeminus contains true trophic nerves, which there form a fasciculus, and that the absence or occurrence of trophic disturbances is conditional on their implication in the same morbid process which has caused the anæsthesia. The solution of this question, however, must be left to experimental physiology and pathology, and we shall here only add that this trophic disturbance ofthe peripheric portion of the trigeminus is wont to be occasioned by only one lesion, whilst in anæsthesiæ of the trigeminus of centric origin this lesion is wanting.

To all appearance the symptoms accompanying anæsthesia present much more of an accidental character than those associated with neuralgia; for the most part they are not directly dependent on the anæsthesia, yet their presence and grouping may afford much aid in the discovery of the localization and nature of the morbid processes to which the anæsthesia is due. An examination, therefore, into the nature of these processes is a matter of no slight importance.

Symptomatology of the Several Forms of Anæsthesia.

We shall here briefly characterize some of the more frequently recurring forms of anæsthesia, omitting such points as may be discussed more elaborately in other parts of this work.

Traumatic Anæsthesia.—The immediate cause of this form of anæsthesia is a wound of some kind or other, such as that produced by gun-shots, contusions, dislocation, etc., or a mechanical compression, as that produced by abscesses, cicatrices, etc. In such cases, the anæsthesia is generally total, and is always accompanied by well-marked motor, vaso-motor and trophic disturbances, as well as by more or less acute excentric pains. The course of the affection is usually very slow, and union generally begins to take place at a rather late period; as a rule, however, it takes place earlier in the case of sensory than in that of motor nerves. In many nerve regions, as, for instance, in those of the median nerve and rami digitales, sensibility returns very soon after the injury, probably in consequence of the presence of communicating branches.

Rheumatic Anæsthesiæ.—Catching cold, a thorough wetting, and similar conditions, are the causes of this form. The anæsthesia is usually preceded by rheumatoid, neuralgiform, or true neuralgic pains, and, in general, is not very marked. Perception of pain, when sharp irritation is applied, is usually preserved. Sometimes, where mixed nerves are affected, there are slight motor disturbances, but symptoms of vaso-motor and trophic disturbances are rare ; herpes may, however, occur. The course is usually rapid, and the termination favorable.

Anæsthesia of Laundresses.—The patient here complains of a feeling of numbness, formication, and a peculiar stiffness in both hands and forearms, but seldom of acute pain. At the same time there are the usual symptoms of vaso-motor cramp, and the hands are pale and cold. The patient, as a rule, is anæsthetic in these parts for all the various kinds of sensation, but the anæsthesia is usually incomplete, so that severe irritations of all kinds can still be felt. Motor disturbances are either altogether absent, or they are of no importance. The affection is obstinate, and often persists long after the occupation has been given up. (It may be classed partly with the foregoing, and partly with the following form.)

Vaso-motor (*Ischæmic*) *Anæsthesia.*—This is usually an incomplete but general form of anæsthesia, exhibiting considerable fluctuations in intensity. The diagnosis is based upon the following circumstances : The fluctuations coincide with those of vaso-motor cramp of the smaller cutaneous vessels ; the anæsthesia is often preceded and accompanied by coldness and pallor, and often also by cyanosis of the skin and smallness of the pulse ; and, lastly, warmth applied externally lessens, while cold augments, the anæsthesia. The course varies in different instances, but is usually rapid and favorable.

Neuritic Anæsthesia.—There is nothing distinctive in the characters presented by this form. It is generally incomplete, and accompanied by well-marked neuralgic symptoms, but seldom by any disturbance of the motor functions. Herpes, on the contrary, is of frequent occurrence. The pathognomonic symptom is the evidence of neuritis in the nerve. This holds also for the anæsthesiæ caused by lepra, neuromata, and other tumors. The diagnosis essentially rests on the proof of the existence of these pathological products.

In the same way, in cases of *toxic anæsthesia*, evidence of pre-existing poisoning is of great importance in determining the diagnosis. This is most readily obtained in cases of chronic poisoning by lead, in which, however, the anæsthesia is seldom of high degree. Anæsthesia from ergotine poisoning generally attacks the fingers and toes at the same time, and is associated with a considerable amount of vaso-motor disturbance; in severe cases the extremities mortify.

Anæsthesiæ following certain acute diseases may be of the most various kinds. After typhoid fever the anæsthesia is often confined to the area of distribution of a single cutaneous nerve, or extends over the area supplied by larger mixed nerves, and is then generally associated with motor disturbances; or it may be of spinal, and sometimes even of cerebral origin. After diphtheria, anæsthesia of the pharyngeal muscles is very frequent, a symptom that is not at all uncommon even after simple inflammatory angina. Moreover, very widely diffused forms of anæsthesia occur, which generally, however, appear to have a central origin.

Hysterical anæsthesia may present very different forms. It may either be local or widely extended over the whole body; it may be confined to one extremity, or appear in an hemiplegic or paraplegic form; it may be partial (most commonly as analgesia) or total, and may attack the mucous membranes (pharyngeal and laryngeal mucous membranes, vagina and vulva) just as well as the skin. Its real nature is as a rule established by other evidence of hysteria. (See the chapter on this subject.)

Syphilitic anæsthesia (Fournier), which chiefly presents itself in the form of analgesia, and occurs only on particular parts of the body (as the backs of the hands, mammæ, etc.), has already been briefly described.

Spinal anæsthesia is one of the commonest forms, and is usually recognized by its affecting the lower extremities on both sides; it may also, however, occur on other isolated parts of the body, as in the area supplied by the ulnar nerve, in tabes; in that supplied by the fifth nerve, and on the trunk. It may be either a partial or a total, a complete or an incomplete, sensory paralysis. The concomitant symptoms of lesion of the spinal cord, as pareses and paralyses (chiefly of a paraplegic form), ataxia, sensation of constriction, vesical and sexual debility, will alone enable us to arrive at a conclusion in regard to the seat of disease. (For further information see the section on Diseases of the Spinal Cord.)

Cerebral anæsthesia can only be easily recognized when it attacks one side of the body; but it often appears to be confined to single parts of the skin, to the districts supplied by particular nerves, or to one extremity, and the diagnosis can then only be established from a consideration of the accompanying cerebral symptoms (motor paralyses, disturbances of the senses, implication of the cerebral nerves, vertigo, diminished intelligence, impaired memory, etc.), but in most cases this is accomplished without any special difficulty. Anæsthesia may accompany either limited or diffused disease of the brain; it does not, however, in general attain a high degree of intensity, and shows a strong disposition to improvement; partial paralysis of sensation may occur, but it is rare. The great variety in the modes of supervention of cerebral anæsthesiæ admits of easy explanation when the wide separation between the sensory, motor, and vaso-motor nerves in the brain, and the numerous forms of diseases by which they may be attacked, are borne in mind. Analgesia is of remarkably frequent occurrence in the psychoses; and simple local anæsthesia is also often present. (For further details on these points we must refer to the chapters specially devoted to these subjects.)

Amongst the anæsthesiæ of peripheral nerves, ANÆSTHESIA OF THE TRIGEMINUS alone requires a more detailed description. It is relatively frequently observed as an isolated affection, and is for the most part a dangerous disease; besides its practical importance, it possesses considerable physiological and pathological interest.

This form of anæsthesia often results from inflammation, atrophic changes, degenerations, tumors, etc., affecting the intracranial part of the trigeminus, and especially the ganglion Gasseri; or from pathological changes affecting the neighboring parts, as the bones, vessels, and membranes of the brain, etc., and extending to the trigeminus; or from injury by fractures of the skull, wounds of the head, operations, etc. The peripheric anæsthesiæ of the trigeminus are due to these or to similar causes, but the fibres in their central course may also be affected in apoplexy, tumors, atrophic processes, sclerosis of the brain, the result manifesting itself as anæsthesia.

The extent of the anæsthesia will of course vary according to the seat of disease, being either limited to a single branch or principal trunk, or affecting the whole region of distribution of the trigeminus. In the latter case, one side of the face, part of the ear, the skin of the temple and forepart of the head, are deprived of sensation. The conjunctiva and cornea, the nasal and oral mucous membranes, the tongue, gums, and a part of the pharynx, are all rendered more or less completely insensitive on the affected side. We are not aware if partial sensory paralysis of the face may occur. When the patient puts a goblet to his lips, it gives him the impression of being broken; he does not feel on the anæsthetic side when he has anything in his mouth, or even when a foreign body enters the eye, etc. Excentric pain not infrequently occurs amongst the concomitant symptoms in the form of a violent tic douloureux. The movements of mastication are sometimes disturbed and sometimes unaffected, which is dependent upon whether the motor root (portio minor) of the fifth is or is not implicated. Those facial muscles that receive their nervous supply from the facial nerve, remain perfectly capable of performing all voluntary movements; yet the movements of the anæsthetized half of the face are not infrequently slow and imperfect. No reflex actions whatever can be called forth when the anæsthesia is peripheric. The skin of the face appears bloated and often of a bluish-red color, and cool; sponginess of the gums, ulcers of the mucous membrane of the mouth and nose, and hemorrhage from these parts, are all symptoms that have been observed; injuries of the anæsthetic mucous membrane lining the cheeks often arise from biting it, and these heal with difficulty, and often lead to the formation of unhealthy ulcers. The ophthalmic symptoms are particularly noticeable. The acuteness of vision undergoes diminution, partly owing to the failure of the lachrymal secretion, and partly in consequence of secondary trophic disturbances and hyperæmia; the cornea becomes cloudy and then ulcerated, and perforation may occur, the eye perishing from atrophy; this, however, has already been mentioned. The sense of smell is only impaired in consequence of the dryness of the Schneiderian mucous membrane. Simple sensory irritation, however, like that which may be produced by ammonia and snuff, is no longer capable of exciting reflex actions. The hearing usually remains unaffected, providing the tensor tympani is not paralyzed by the disease, in which case a slight disturbance of the hearing may be produced. The sense of taste, on the contrary, is almost always considerably impaired. The perception of taste is lost throughout the anterior two-thirds of the anæsthetic side of the tongue. It is well known that the sense of taste in these parts of the tongue is due to the lingual nerve; the gustatory fibres, however, for the most part, run from the facial through the chorda tympani. As, however, there are a number of well authenticated cases (as, for example, those reported by Hirschberg, Guttmann, Kocher, Hippel, and others) in which complete anæsthesia of the trigeminus was present with complete integrity of the facial, yet in which there was loss of taste on the anterior half of the tongue, it is probable that the gustatory fibres of the chorda in reality originate from the trigeminus. When, therefore, this nerve is damaged at any part in its course where it contains gustatory fibres—which, from the preceding observations, appears to be the case at the base of the skull—paralysis of the sense of taste results. We shall return to this question, which is important in so many respects, in speaking of neuroses of the gustatory nerves and paralysis of the facial (see the chapters on this subject). No statements have been made in regard to the behavior of the salivary secretion in trigeminal anæsthesia.

Such is a brief sketch of anæsthesia of the fifth nerve, as it presents itself partly as an isolated affection, and partly as a symptom in various diseases of the brain and base of the cranium. The seat of the disease and its peripheral or central origin can be easily ascertained by a little attention to the parts affected by anæsthesia, to the presence of motor, trophic, and sensory disturbances, and to the existence or non-existence of functional disturbances of the nerves at the base of the brain, or of the brain itself. (For additional information regarding the diagnosis, see Romberg).

Anæsthesia of the several spinal nerves requires no particular description; its seat and extent depending upon their anatomical distribution, whilst its etiology and symptomatology may be deduced from the statements that have just been made.

Course, Duration, and Terminations.

An account of the course, duration and terminations of anæsthesia may be comprised in a few words: these all depend almost entirely upon the nature and curability of the primary disease. Anæsthesia may be either sudden or more or less gradual in its mode of commencement. In favorable cases, after the disturbance has reached its acme, gradual improvement commences, and recovery finally takes place; as, for instance, in many central anæsthesiæ, and in the rheumatic, ischæmic, and traumatic forms. In unfavorable cases the disease becomes either gradually or suddenly worse, advancing from partial to total, and from circumscribed to diffused anæsthesia; serious trophic disturbances then supervene, namely, extensive bed-sores, which may lead to death through exhaustion or from pyæmia. Yet recovery is still possible in states of advanced anæsthesia. Everything here depends on the nature of the primary disease; but to enter minutely into all the different courses the disease may take, would be to recapitulate the whole pathology of the central nervous system. It often happens also that when amendment has commenced, one or other of the sequelæ of the disease remains behind: tactile sensibility remains for a long time dull, and is clearly more diminished by cold than in healthy parts of the skin. Sometimes particular kinds of sensation are wanting for a longer period than others, or are permanently lost, and in this manner a kind of incomplete cure can be effected.

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Diagnosis.

In the *diagnosis* of sensory paralysis, several different points have to be considered. In the first place, we should determine clearly the extent and degree of the disease ; this can be readily done by aid of the several methods of investigation given above.

In the next place, it is necessary to discover the precise *seat of the primary disease*, the most important point being to determine whether the anæsthesia is of peripheric or of central origin. The principal points, as may be gathered from the foregoing description, are the extent and the degree of the anæsthesia (partial sensory paralysis occurring almost exclusively in central, scarcely ever in peripheral disease), the accompanying motor, vaso-motor, and trophic disturbances, the mode in which reflex acts are performed, the existence or non-existence of spinal and cerebral symptoms, the evidence of the existence of different causes, etc. The diagnosis between true peripheral anæsthesia and anæsthesia of the peripheral conducting organs, on the one hand, and between true central anæsthesia and anæsthesia of the cerebral conducting fibres, on the other hand, will in most cases be rendered evident by a study of the details of the affection. It is not, however, always possible to make the diagnosis with certainty.

Finally, the diagnosis should include the determination of the causes of the anæsthesia, and the pathological and anatomical changes that have produced it. No detailed statement can be made on this point; and the requisite information must be gained by a careful consideration of the several symptoms, and by duly weighing them in the light of the principles of special pathology. In this manner it is often possible to make an exact diagnosis ; but where that cannot be done, we must rest satisfied with vague suppositions in regard to the seat and pathological anatomy of the disease, and this is especially the case in those large groups of anæsthesia which depend on the so-called impalpable trophic disturbances (as hysteria, the different forms of poisoning, rheumatism, etc.).

Prognosis.

The *prognosis* of anæsthesia depends almost entirely upon the cause, or rather upon the seat of the disease. A favorable termination may be expected in rheumatic, ischæmic, hysterical, and syphilitic anæsthesiæ, and in the forms induced by the peripheral action of cold or corrosives. So also where the compression causing the anæsthesia is moderate, a favorable prognosis may be given, if the compressing cause can be removed; but if, on the contrary, the compression has been so strong as to produce degeneration of the nerve, the prognosis is less favorable, and a long period must at least elapse before the cure is accomplished. This is still more true of cases of entire division of the nerve, for degeneration then always occurs, and everything depends upon whether the divided ends of the nerves are in a favorable or unfavorable position for union and regeneration. In the former case, recovery takes place after a certain time (generally several months), whilst in the latter a period of months or years may be required, or the affection may remain permanently incurable. Anæsthesia after acute diseases runs for the most part a favorable course, as do also those arising from toxic influences. The prognosis in spinal and cerebral anæsthesiæ is usually, though not always, identical with that of the primary disease; in cerebral forms (those due to apoplexy or embolism) it may not unfrequently be noticed that the sensory paralysis soon disappears, whilst the other symptoms persist for some time.

In general the prognosis in cerebral anæsthesiæ is more favorable than in the spinal forms. If the latter be complete and well marked, and if they be at the same time accompanied by serious trophic disturbances, the prognosis is generally bad. (For further information, see the chapters on diseases of the spinal cord in another part of this work.)

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Treatment.

The causal indications must first be attended to. In a large majority of cases direct treatment, or the treatment of symptoms, proves unsuccessful, if the cause of the anæsthesia have not been removed, whilst it often happens that the cure of the primary disease suffices to cause the removal of the anæsthesia. It would lead us too far here to give even the outlines of the therapeutic measures that may be required to fulfil the causal indications ; it is certain that the most diverse surgical proceedings, numerous internal and external remedial measures, and particularly electro-therapeutics, hydro-therapeutics, and balneo-therapeutics, may play an extensive and important rôle in the treatment. Particular attention must in many cases be directed to the primary diseases of the central nervous system.

If, however, no cause can be demonstrated, or if, after the removal of the cause, the anæsthesia still remains more or less complete, or, if it seem advisable, in addition to the causal treatment, to attack simultaneously the symptom of anæsthesia, then the *direct treatment* of anæsthesia must be commenced, and that symptom becomes itself the object of appropriate therapeutic measures. Such direct treatment is often indispensable to effect complete recovery, and it may frequently be noticed that even after the removal of the cause, and after the cure of the primary disease, the anæsthesia only yields to direct treatment. Where, however, a cause exists which the means at our disposal do not enable us to remove, nothing can be expected from the direct treatment of the anæsthesia, and it may be omitted as superfluous.

The principal aim of this direct treatment is the *re-establishment of the diminished or obliterated irritability and conductivity of the sensory apparatus.* This object will be best attained by applying properly graduated irritants to the periphery, which, on the one hand, are capable of exalting the diminished irritability, and, on the other, of overcoming any obstacles to the conductivity of the fibres that may be present.

By far the most effective of all the remedies that can thus be applied is indisputably *electricity*, a means of excitation that may be employed in the most various degrees of intensity, and can be everywhere applied with the utmost facility, and without being followed by any unpleasant secondary effects. The methods best adapted to this object are (not referring to the very numerous and important methods of employing electricity in the treatment of the primary disease [such as spinal and cerebral diseases, neuritis, vaso-motor disturbances, cramp, etc.], methods which are mentioned in the chapters of this work devoted to these subjects, and in the elementary books on electro-therapeutics):—

a. Faradization of the skin, by means of the electric brush, or, if a less powerful irritant is required, by means of moist electrodes. The induction current is best obtained from the secondary coil, and the cathode (of the opening current) may be applied as the cutaneous irritant, whilst the anode is placed on some indifferent part; the current should, in general, be of considerable strength, and the irritation should last for a couple of minutes.

b. Galvanization of the skin is best effected by moving the cathode to and fro over the thoroughly moistened skin, a strong current being used for the purpose. The anode must be placed over the seat of the disease, or upon some indifferent part. In many cases energetic coincident irritation of the nerve trunks is useful. The employment of the electric brush in connection with the cathode of the galvanic current is less advantageous; when thus used, the current must be of extraordinary strength, and is then likely to produce slight cauterizations of the skin. The application should last for the same length of time as when the faradic current is used, and the frequency of application should be regulated on general principles. In curable cases improvement usually occurs after each sitting, and lasts for a progressively longer period, till at last it results in permanent recovery.

Many other cutaneous irritants act in the same way as electricity, and particularly the spirituous, ethereal, and other stimulating embrocations (opodeldoc, spirit of camphor, oil of turpentine, oil of mustard, spirit of ants,[1] spirit of wild thyme, tincture of cantharides, veratria ointment, etc.). Still more intense effects can be obtained by urtication, by the alternate application of hot and cold shower-baths, dry carbonic acid baths, etc. Warm baths, thermal air baths, and simple friction, act more gently. Türck has already demonstrated the fact, that the lesser degrees of anæsthesia can be removed by friction alone, and hence the much-vaunted success of many embrocations may be due in part to the mechanical irritation of the rubbing.

The more energetic counter-irritants, such as vesicants, ointments producing pustules, moxæ, and the actual cautery, should only be employed when there are very distinct indications (usually causal) for their use; but we expressly raise a warning voice against their indiscriminate application to anæsthetic parts, which are always liable to serious trophic disturbances.

Internal remedies are generally prescribed exclusively in accordance with the causal indications. Strychnine has been recommended as a specific remedy for anæsthesia; and other nervines, such as musk, castor, valerian, arnica, etc., have also been recommended, but their action is exceedingly doubtful.

In anæsthesia of the trigeminus, the treatment appropriate to the presumed primary disease must be adopted. The application of the galvanic current is one of the most important means that can be employed in this affection, but great caution is required in its use on account of the proximity of the brain. A protective bandage over the eye constitutes the best remedy for neuroparalytic ophthalmia. Trophic disturbances of the skin should be treated in accordance with the ordinary principles of surgery.

The *dietetic treatment* (mode of life, food, etc.), should be regulated by the cause to which the disease is referable. The prophylactic protection of anæsthetic parts against serious trophic disturbances is deserving of particular attention. The appropriate remedies for it are: suitable position and support of the parts, avoidance of any constant pressure, the most scrupulous cleanliness, and ablutions with cold and spirituous fluids. The occurrence of trophic disturbances, however, cannot always be successfully and permanently prevented.

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[1] Ants, recently gathered and bruised, 10 parts; alcohol, 15 parts; water, 15 parts. Macerate for two days, then distil off 20 parts.—*German Ph.*

Muscular Anæsthesiæ.

Nasse. Zeitschrift für psych., Aerzte, 1822, Heft II.—Charles Bell, physiol. u. pathol. Unters. des Nervensyst. Deutsch v. Romberg. 1832, p. 175.—*O. Landry,* Sur la paralysie du sentim. d'activité muscul. Arch. génér. 1852.—Gaz. d. hôp. 1855. —*Duchenne,* Electrisat. localisée. II. edit. pp. 389 u. 424. 1861.—*E. Leyden.* Ueber Muskelsinn u. Ataxie. Virch. Arch. Bd. 47. 1867.—*Bernhardt,* z. Lehre vom Muskelsinn. Arch. f. Psych. u. Nervenkrankh. Bd. III. 1872. Consult also the text-books of *Romberg, A. Eulenburg,* and others, and also the manuals of physiology.

In consideration of the relatively small practical importance which this subject possesses at the present time, we shall here only shortly notice the so-called *muscular anæsthesiæ* and allied phenomena. It may scarcely appear judicious to discuss these symptoms of disease in a work devoted to diseases of the peripheral nervous system, since the very existence of peripheral nerves for muscular sensibility has not as yet been anatomically established. Moreover, muscular anæsthesiæ are so essentially symptoms of disease of the central and especially of the spinal nerves, that we must reserve a more detailed consideration of the subject for the sections devoted to the several forms of centric disease. A brief mention of the subject, however, in this place is scarcely avoidable.

The anatomical paths for centripetal excitations proceeding from the muscles are quite unknown ; by many observers, indeed, their existence is entirely denied, and it is maintained that all sensations of this kind are transmitted through sensory fibres traversing the muscles in their passage to neighboring organs (fasciæ, etc.). This much only appears to be certain, that if there be any sensory muscular nerves, they leave the spinal cord with the posterior roots, and in their further course join the motor nerves and run together with these to the muscles. It is only lately that Arndt[1] found in the muscles

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[1] Arch. f. mikrosc. Anat. IX. p. 481.

(in all classes of animals) fine nerve fibres which twine around the sarcolemma, and appear to end in it, and which he regards as the sensory nerves of muscle. Rauber holds that the corpuscles of Vater, found in the sheaths of the muscles, in the periosteum, and in the vicinity of the joints, perform some at least of the functions, which are generally attributed to the so-called "muscular sense."

The *physiological* investigations which have been made in regard to the sensibility of the muscles have left many hiatuses to be filled up, and much that is obscure ; and no definite agreement has been arrived at even in reference to the principal points.

It cannot be denied—quite apart from the existence of anatomically distinct nerves and of the termination of these nerves in the muscle itself or in the adjoining tissues—that certain sensations are experienced which arise in the muscles, and which stand in the most intimate connection with their function. These sensations, although quite independent of those communicated by the skin, have still a certain degree of similarity with the latter. These sensations partly resemble those of common sensation ; such as belong to this class being named *muscular sensibility*—and partly resemble those of tactile sensibility, as exhibited in the estimation of weights, and of different conditions of resistance and contraction ; these are included under the term "muscular sense," or "*sense of muscular effort.*" By the term *muscular feeling* or *sensibility* is to be understood the dull and indefinable sensation which is perceived in the muscles themselves when energetic contractions are made, and which may increase to positive pain (as in cramp); also the similar sensation which is experienced when the muscles are exposed to faradic excitation, and which augments in its intensity in proportion to the strength of the current applied till violent pain is felt, and which can easily be distinguished from the pain caused coincidently in the skin by faradisation (*electro-muscular sensibility*) : the sensation of fatigue, which is experienced in the muscles after protracted use, must also be included under this head ; and, lastly, we must mention the sensitiveness to firm pressure which healthy muscles exhibit when they are squeezed, or when they receive a blow, a sensation which can easily be distinguished from the coincident sensation in the skin. Again, the means of tormenting, in favor with boys, that, namely, of violent pinching of the muscular biceps brachii to produce severe pain (a procedure which may be counteracted by energetic contraction of this muscle), sufficiently demonstrates the non-participation of the skin in the pain. The muscular sense can be tested either by external pressure, or, what is far better, by faradisation of the muscles. By this means, if the current be sufficiently strong, the characteristic dull sense of contraction is distinctly felt, and by measurement of the distance separating the two coils a comparable measure of the intensity can be obtained.

The sense of muscular effort enables us, in the first place, to estimate differences of weight and pressure, and indeed more delicately than is possible by means of the mere sense of pressure on the skin alone. Thus, according to E. H. Weber, differences in weight, bearing the same relation to each other as 39 : 40, can be distinguished by the muscular sense. This sense enables us to estimate the amount and extent of the muscular contraction, and the degree of resistance opposed to it,—that is to say, to estimate the force applied to overcome a given resistance (sense of force or effort). It is probable, also, that the muscular sense participates, to a certain extent, in the power we possess—even with closed eyes—of knowing exactly the position of our members, and of being able to alter their position in a determinate manner; it also has some share in our capability of maintaining under the same circumstances the equilibrium of the body. It is highly probable, however, that many other sensory impressions, such as those derived from the surfaces of the joints, from the ligaments, tendons, fasciæ, external skin, etc., co-operate in this faculty. As these several factors cannot easily be distinguished from each other, and as disturbances in them are produced by these same methods of investigation, we shall consider them as belonging to the muscular sense.

For testing the muscular sense the patient should be made to lift various weights, and to form an estimate of their differences (care being taken that no assistance to the judgment be afforded by the sense of pressure on the skin). The weight should be placed in a cloth and suspended from the limb to be investigated, or it may be so held in the fingers that it is supported rather by friction than by pressure. The determination of the smallest perceptible weights can thus be made, and by comparing the results of such experiments with those obtained in healthy persons, any pathological condition of the muscular sense that may be present can be ascertained. Such tests of the sense of force may also be made by means of the dynamometer. The patient may also be made to move the limb into certain prescribed positions, with closed eyes, to raise it to a certain height, and to put it in the same position as the passively moved opposite limb ; he may be directed to touch a part of the surface of the body which has just been touched, or to take hold of a ticking watch or a ringing bell held before him. Every experiment of this kind must be made with closed eyes. This can all be executed with remarkable precision by healthy persons, but it is often astonishing with how little accuracy the movements required can be performed in disease.

This is not the place to enter into the still pending physiological discussion upon the real existence of the sense of muscular effort and of muscular sensibility ; that is to say, of the functions dependent on sensory muscle-nerves. The questions here at issue are in the highest degree difficult of solution, but it is a matter of no particular importance at present which way they may eventually be decided. Important names declare—though with many restrictions and differences—for the existence of muscular sensibility : as Charles Bell, Romberg, Bernard, Landry, Duchenne, E. H. Weber, Leyden, and others ; whilst others, as Spiess, Lotze, Remak, and Schiff, deny the existence of either form. Only lately Bernhardt (loc. cit.) has transferred the seat of the so-called muscular sense to the parts surrounding the muscles, and regards the so-called sense of force as a function of the mind, which is quite independent of centripetal impressions originating in the muscle itself. The remarkable observations, made by Weir Mitchell on stumps after amputation, possess a very high degree of interest in relation to this question.[1]

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[1] Injuries of Nerves, Philadelphia, 1872, p. 353 et seq.

He found that, after amputation of the upper arm, for instance, the patient retained a distinct feeling of the position and movement of the amputated hand,—which is undoubtedly in favor of the central origin of these sensations. He also found—what is still more wonderful—that faradisation of certain of the nerve trunks in amputated limbs renewed the feeling of motion and change of position in the now absent parts, just as if they were still attached,—which is again in favor of the excitation being centripetally conducted. These facts deserve attentive consideration. It is not our business here to decide the pending physiological question ; we have simply to bear in mind the practical fact, that, in consequence of pathological conditions, the sensations that we term muscular sensibility and muscular sense, can be modified in one direction or another, independently of cutaneous sensibility ; and it is at present of very little importance whether we do or do not know accurately the mode in which these sensations arise and are brought to our consciousness. It is only repeated and careful pathological observations that will in time throw some light on these points.

Anæsthesia of Muscular Sensibility.—This is recognized most easily by electrical examination ; for, when this is applied, none of that dull feeling of contraction, which is ordinarily perceived in faradisation of healthy muscles, is perceived ; nor is there any sensitiveness of the muscle when heavy pressure is applied. This kind of anæsthesia may occur either with or without cutaneous anæsthesia ; it is, however, usually accompanied by muscular paralysis ; at the same time, the sense of muscular effort may remain intact. This anomaly occurs most frequently in hysterical cases, and Duchenne has pointed out as characteristic of this paralysis that the electro-muscular contractility is preserved, whilst on the contrary, electro-muscular sensibility is abolished. This, however, is not universally true. Careful researches have shown that anæsthesia of muscular sensibility may exist in many other forms of paralysis.

Anæsthesia of the Sense of Muscular Effort.—This form of anæsthesia is manifested by a diminution or loss of the capability of perceiving differences of weight, or indeed of recognizing small weights by muscular contraction ; in other words, the minimum resistance which it is possible to recognize increases in amount, and the same increase will be observed in the minimum differences that can be recognized between different weights. It is evident that this can only be clearly established when a certain degree of mobility is preserved ; no test of the sense of muscular effort can be applied when the muscles are completely paralyzed. But this form of anæsthesia is also rendered evident by loss of the feeling of the equilibrium of the body, by inability to determine, with closed eyes, the position of the limbs, or the extent of any movements that may be performed, etc. ; in addition, however, there is something more than simple loss of the sense of muscular effort, since other sensory parts participate to some extent in the above-named actions. The loss of the sense of muscular effort may also occur in hysterical patients, but in these the cutaneous sensibility and the muscular sensibility are generally both coincidently paralyzed. Anæsthesia of the sense of muscular effort is most frequently observed in gray degeneration of the posterior columns of the spinal cord (tabes dorsalis) ; here, however, the symptoms appear to present great variety ; for instance, the patient can, in many cases, still correctly recognize differences of weight, whilst the minimum-resistance which he is able to distinguish will be found to have materially increased ; the feeling of equilibrium and the knowledge of the position of the limbs are very often no longer normal ; the electro-muscular sensibility, however, is generally preserved ; the tactile sensibility may be modified in various ways. For further information the reader is referred to the description of tabes dorsalis.

There is little to be said in regard to the method of *treatment* that should be adopted in cases of muscular anæsthesia ; as a rule, the treatment must be that which is appropriate for the primary affection. For the most part no particular treatment is required, but if it be desired to institute some form, nothing better can be recommended than the direct or indirect faradisation or galvanization of the muscles.

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NEUROSES OF THE NERVES OF SPECIAL SENSE.

These constitute an obscure and not easily definable territory in pathology. The phenomena that were formerly included under this head are gradually becoming more intelligible, and the increased knowledge we possess of other diseases of the apparatus of special sense is perpetually narrowing the area of the so-called sensory neuroses. The pathology of the two higher nerves of special sense—the optic and the auditory—has made the greatest advances. As the nerves of these last belong to a special department, which is represented by a rich literature of its own, we must refer the reader to the manuals devoted to the eye and ear. We shall here only discuss the neuroses of the nerves of taste and smell.

1. Neuroses of the Gustatory Nerves.

Henle, Lehrbuch der Anatomie, Bd. III. 2. Nervenlehre, 1873.—*Romberg*, Nervenkrankheiten 3. Aufl., Bd. 1. pp. 148 and 301. 1853—*A. Eulenburg*, funct. Nervenkr. p. 292.—*Stitch*, Beitr. z. Kenntn. d. Chorda tymp. Annal. d. Charité, VIII. 1857.—*Lotzbeck*, Deutsch. Klin. 1858, No. 12.—*Ziemssen*, Virch. Archiv. XIII. p. 376. 1858.—*Neumann*, Electr. als Mittel z. Unters. des Geschmacksinns u. über Geschmacksfunct. d. Chorda. Königsb. med. Jahrb. IV. 1864.—*Moos*, Störung des Geschmacksinns durch Druck auf die Chorda tymp. Centralbl. 1867, No. 46, Arch. f. Augen- und Ohrenheilk. Bd. I. 1. p. 207.—*M. Schiff*, Neue Unters. über d. Geschmacksnerven d. vord. Theils der Zunge. Molesch. Untersuchungen, Bd. X. p. 406. 1867.—*Schiff*, Intorno ai nervi del gusto, etc. Morgagni, 1870.—L'Imparziale, XI. 1871, No, 15; u. XII. 1872, No. 14.—*Lussana*, Recherches experim. et observ. pathol. sur les nerfs du gout. Arch. de physiol. norm. et path. 1869.—*Lussana*, Sui nervi del gusto. Gazz. med. Ital. Prov. Venet. XIII. No. 42—46, 1871.—*Lussana*, Sur les nerfs du gout. Arch. de physiol. norm. et path., 1872.—*Prévost*, Note rélat. aux fonct. gustat. du nerf. lingual. Gaz. méd. d. Par. 1869, Nos. 36 u. 38- *Kocher*, *Hirschberg*, *Guttmann*, Fälle von Trigeminuslähmung. Berlin klin. Woch. 1868, Nos. 10 u. 11; 48 u. 49; 51.—*A. Wernich*, Beitr. z. d. Parästhes. des Geschmacks. Arch. f. Psych. u. Nervenkr. II. 1869.—*Erb*, 2 Fälle von traumat. Facialparalyse. Arch. f. klin, Med. VII. p. 246. 1870.—*Steiner*, Rückenmarkshyperämie; Unters. über d. Chorda tymp. bei doppelseit. central. Facial-paralyse. Arch. d. Heilk. XI. 1870.—*Guttmann*, seltner Fall v. Hysterie, etc. Berl. klin. Woch. 1869, No.

28 u. 29.—*Tillmanns*, über Faciallähmung, bei Ohrkrankh. Diss. Halle, 1869. —*Althaus*, z. Phys. u. Path. des Trigem. Arch. f. klin. Med. VII. p. 563. 1870.—*Seeligmüller*, neuropathol. Beobachtung, Halle, 1873. 4. Beob.

The anatomy and physiology of the gustatory nerves still constitute a fruitful field for discussion ; there exist at least considerable differences of opinion amongst the principal inquirers on several essential points. The following account of our present knowledge of these subjects is an endeavor to give, as far as possible, those facts which may be regarded as either well established, or which are in the highest degree probable ; it may serve to complete what has been said, in speaking of trigeminal anæsthesia, in regard to disturbances of the gustatory functions, and we shall hereafter have to dwell upon it more at length in speaking of paralysis of the facialis.

The principal organ of the gustatory sense is the tongue ; but the sensations of taste are also perceived by part of the soft palate, by the arches of the palate, and by the walls of the pharynx. This much, at least, is certain, that the root of the tongue, and the pharyngeal tissues on the one hand, and the anterior half of the tongue on the other, receive their gustatory fibres from different nerves.

It has been satisfactorily established by numerous experiments on animals, made by Schiff, Lussana, Inzani, and others, and it is undoubtedly true also in the case of man, that the gustatory fibres distributed over the posterior part of the tongue, the palate, and the walls of the pharynx, proceed from the glosso-pharyngeal nerve. The anterior two-thirds of the tongue and the tip of the tongue receive their gustatory fibres from the nervus lingualis, as is shown by the fact that section of this nerve near its entrance into the tongue completely abolishes the sense of taste in these parts. This is sufficiently proved both by experiments on animals, and by equally trustworthy observations on man (resection of the lingualis for the relief of neuralgia [Busch, Inzani, Von Graefe, Vanzetti]). Moreover, it has been found that section of the *chorda tympani* (or paralysis of the facialis at any point where it contains the fibres of the chorda) also produces paralysis of the sense of taste, or at least retardation in the rapidity of perception of the sense of taste (Bernard, Schiff, Lussana, Neumann, Stich, Tillmanns, and others) in the anterior part of the tongue. It was thus established that the chorda tympani also contains gustatory fibres, and the question necessarily arose whether all the gustatory fibres pass from the lingualis to the chorda tympani, or whether a part remains in the lingualis. Schiff, from the results of his experiments, inclines to the latter opinion, since, after section of the chorda tympani alone, as well as after section of the lingualis (above its union with the chorda) alone, he in each instance only observed dullness, but no complete paralysis of the sense of taste. Others, on the contrary (Neumann, Lussana), regard the chorda tympani as the only gustatory nerve for the anterior half of the tongue. In any case, it is certain that the chorda contains a very considerable part, if not all, of the gustatory fibres for the anterior half of the tongue. It is probable, however, that there are great individual differences in this respect; but the farther course of the chorda fibres to the brain is still another matter of dispute. Some experimenters (Bernard, Lussana, Inzani) declare that they pass to the brain in the facialis, and that they are contained in the portio intermedia. The proofs, however, that have been hitherto advanced in favor of this view are as yet insufficient. Section of the nervus facialis at the base of the skull gives equivocal results, whilst paralysis of the sense of taste has not been established with certainty (Stich) in those paralyses of the facial which originate in disease at the base of the skull. In an important case of Ziemssen's, bearing on this point, unfortunately no post-mortem was obtained; there was anæsthesia of half of the tongue on the same side.

Others (particularly Schiff) maintain that the gustatory fibres of the chorda pass from the facial into the trigeminus, and, indeed, into its second division, by means of the nervus petrosus superficialis major or some of the other communicating branches. In the same way it is thought that the few gustatory fibres remaining in the lingualis, at the level of the otic ganglion, pass from the third, through different inconstant communications, to the second division, and from thence to the common trunk of the trigeminus. Schiff's numerous and variously-modified experiments, which seem to surpass, in the precision in which they have been executed and in the clearness of the results obtained, those of other observers, are strongly in favor of this view. Section of the trigeminus at the base of the skull completely abolishes the sense of taste in the anterior half of the tongue, and the same result follows the division of the nervus petrosus superficialis major, the division of all the connections of the spheno-palatine ganglion, and, lastly, the extirpation of the spheno-palatine ganglion itself. The results of his experiments have, however, led Prévost to oppose this view. He found no disturbance of the sense of taste after extirpation of the spheno-palatine ganglion. Schiff, in a detailed criticism of these experiments, has proved that the evidence is defective, and has repeatedly established in recent experiments, in which sources of error were carefully guarded against, that extirpation of the ganglion does really annihilate the taste in the anterior half of the tongue. (Morgagni, 1761.)

All these experiments on animals have, it is obvious, very great difficulties to contend with, because it is requisite to show the existence of subjective sensations. Pathological observations on man are consequently of much more importance and value, because here the extent and degree of disturbance can be much more easily ascertained. Of course, those cases only are to be regarded as affording satisfactory evidence which have been subjected to very careful and critical investigation. Amongst pathological conditions appear:—

1. *Cases of complete isolated anæsthesia of the trigeminus* consequent upon disease of the segment lying at the base of the skull), *with coincident abolition of taste in the anterior half of the tongue* (Romberg's case, No. 3, p. 302; von Meyer in Romberg, p. 306; Rigler in Romberg, p. 272; Anstie, Hirschberg, Kocher, Guttmann, Seeligmüller, etc.).—The cases, on the other hand, where the sense of taste is stated to have been preserved in the anterior half of the tongue, with complete anæsthesia of the fifth nerve, are all more or less doubtful. The case of Stamm (in Romberg, p. 308), does not prove anything, as some of the fibres were preserved intact, and, in addition, the perception of sensations of taste was clearly retarded. The case of Bérard, cited in the same place, is also of little value, since both

the trigeminus and the facial nerve were injured, and still taste was preserved (probably, however, only in the glosso-pharyngeal). The case adduced by Romberg himself (p. 312) proves exactly the contrary of what Romberg infers from it. The taste was preserved in the posterior part of the tongue, and, on the contrary, abolished in the anterior and middle parts. The case so often cited by Renzi, which forms the principal means of proof for Lussana, proves nothing, because it is not determined whether all the fibres of the fifth nerve were affected ; it was not even subjected to microscopical examination. In the same way a case of Vizioli's, cited by Lussana (1872), proves nothing positively, as it is not clear what was the real seat of the lesion, and there was no post-mortem. The same objection may be made to the case of Althaus ; the taste was here, in addition, distinctly retarded, and it was only investigated two years after the commencement of the affection, when a decided improvement had taken place in the other fibres of the fifth nerve, and when, in all probability, the glosso-pharyngeal had become vicariously active. It is clear that cases of disease of the fifth nerve with negative results can, in regard to disturbances of taste, only afford positive evidence when it can be ascertained that all the fibres running in the trunk of the trigeminus are damaged, and that there is no central disease. But evidence to this effect has not as yet been adduced. Cases of trigeminal anæsthesia, with positive disturbance of the sense of taste, are of more importance because the evidence of disease confined to that part of the trigeminus which lies in the cranial cavity, concurrent with disturbances of taste, affords a strong probability that that part contains the fibres of taste.

2. *Cases of isolated complete peripheric paralysis of the facial, with abolition of taste on the anterior half of the tongue.* (Neumann, Roux, Cohn, Vizioli, Lussana, M. Meyer, Erb [Case I], Tillmanns, and others.) To these may be added the analogous cases of suppuration of the middle ear, or of caries of the temporal bone, with paralysis and lesion of the chorda tympani. Lastly,

3. *Cases of paralysis of the facial at the base of the skull, above the geniculate ganglion, without injury to the sense of*

taste.—Stich was unable to discover either in the literature of the subject, or in the records of "his own large experience" any instance of basal paralysis of the facial, in which the taste was impaired. (See also Erb's Second Case.) A case of this kind well worthy of notice has been recorded by Wachsmuth (Bulbär paralysis, p. 321). In this instance both facial nerves were found to have undergone complete fatty degeneration in their course along the base of the skull, yet the taste was preserved in the anterior half of the tongue. The fibres in the nervus petrosus superficialis major were intact.

In view of these facts, which constitute the rule in by far the majority of cases, the only conclusion that can be arrived at is that *the greater part of the fibres ministering to the sense of taste pass from the nervus lingualis into the chorda*, *and by means of the latter into the facialis*, *but finally return again into the trigeminus.* The different courses pursued by the nerves in their return are certainly not yet exactly known, but they may be the following:—1. Fibres may run from the geniculate ganglion through the nervus petrosus superficialis major and nervus vidianus to the spheno-palatine ganglion, and then into the second branch of the trigeminus. Cloquet, Hirzel, Hirschfeld, and Leveillé, have, upon purely anatomical grounds, claimed the existence of this connection as the direct continuation of the chorda. 2. Peripheric fibres of the chorda may run in the facial, and then pass through communicating branches to the auriculo-temporal, from thence to the otic ganglion, and from thence to the spheno-palatine or Gasserian ganglion. The presence of these fibres would afford an explanation of those cases recorded by Stich and Lotzbeck in which division of the facial nerve below the stylomastoid foramen caused retardation of the sense of taste, a result which is certainly not of constant occurrence, as I have recently satisfied myself in a case of extirpation of the parotid. 3. Direct communication may exist between the chorda and the otic ganglion, etc. 4. Fibres may pass from the geniculate ganglion through the tympanic plexus to the lesser superficial petrosal nerve, to the otic ganglion, etc. There are still several other means of connection for the fibres of taste which remain in the lingualis, and it is possible that the fibres may run very differently in different individuals; but this is of little importance, the principal point being that the larger portion of the gustatory fibres, supplying the anterior part of the tongue, run, in the first instance, in the lingualis, then in the chorda, and for a short space in the facial, returning ultimately to the trigeminus, with which they enter the brain.

One more possibility may here be mentioned, namely, that fibres of the chorda tympani may run through the otic ganglion, and in the tympanic branch of the glosso-pharyngeal nerve, and pass to the brain in this nerve. This possibility, however, in view of the experiments and pathological observations that have been made on the trigeminus, is not a very probable one; still it may happen, and an explanation of otherwise paradoxical cases would then be afforded.

It is impossible to deny that such cases do occur and have been observed, and it is conceivable that paralysis of the facial nerve and anæsthesia of the trigeminus may exist without impairment of the sense of taste, and, in so far as they are not explicable upon the theory of an incomplete destruction of the nerves affected, they are probably referable to varieties and abnormal conditions, in each case, of the paths pursued by the gustatory fibres in their course to the brain. Lastly, the existence of communications between the lingual and the glosso-pharyngeal nerve in the tongue itself, materially augments the difficulty of drawing correct conclusions from clinical phenomena.

If we now recapitulate the present state of our knowledge in regard to the gustatory nerves, it appears that the base of the tongue, the palate, and the walls of the pharynx, are supplied by the glosso-pharyngeal nerve; the anterior two-thirds of the tongue, on the other hand, is supplied by the lingual; the gustatory fibres of these nerves, however, chiefly, if not altogether, enter the chorda tympani, then run in the facial at farthest only to the ganglion geniculatum, and finally return by various and in many still imperfectly known paths to the second and third divisions of the fifth nerve, in which they ascend to the brain.

Our knowledge is equally inexact both as regards the intra-

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cerebral course of the gustatory fibres and in regard to the position of the perceptive centre of the gustatory sense.

Physiology teaches us that the regions of the tongue which are endowed with the most exquisite gustatory sensibility, are the borders, the tip, and especially the root, but that the faculty is possessed by several parts of the palate, by the gums, and by a part of the walls of the pharynx, and that by the mutual contact and pressure of these parts, the sensations are rendered more acute and distinct. No light has as yet been shed upon the essential nature of the sense, and if the sensations experienced are arranged under different categories, of which bitters, sweets, acids and salines are the chief, these are purely conventional, and by no means exhaust the manifold varieties of sensation which are capable of being recognized.

According to Lussana, the tastes of many things differ considerably, according to whether they are perceived by the fore or the back part of the tongue. Food and tasty substances are particularly well and distinctly perceived by the anterior part of the tongue, whilst the root of the tongue is especially adapted for the perception of the simple special tastes (bitters, acids, alkalies, etc.). No substance can be tasted unless it be dissolved, and the majority of gustatory sensations are accompanied by a very distinct sensation of gratification or loathing, of pleasure or dislike, etc.

For the purposes of pathological investigation, it is sufficient if we confine ourselves to the above-mentioned principal categories of gustatory sensations, and endeavor to ascertain their impairment or disturbance with precision. In such inquiries, however, it is necessary to take certain precautions : thus it is expedient that the patient should put the tongue out, with the mouth widely opened, and that he should keep the eyes closed whilst the sapid substance is being applied on the particular part, the sensibility of which is to be investigated, with a glass rod or small brush, and in very small quantity. As soon as a taste is perceived, a sign is made, and then only is the tongue to be withdrawn and the conclusion arrived at stated. The chief difficulty experienced is in keeping the stimulus perfectly localized or circumscribed, or in other words, in preventing its diffusion over adjoining or other parts of the tongue. After each experiment, the tongue must be prepared for a fresh trial by rinsing the mouth out with water.

For testing *bitter* tastes, it is customary to use solution of quinine, decoction of colocynth or quassia, solution of picric acid, &c. Bitters are most distinctly perceived at the root of the tongue. The quality of *sweetness* is tested by means of solution of sugar, syrup, honey, &c., and is most distinct at the tip of the tongue. For testing *acids*, vinegar, diluted acids, wine, etc., are selected, and these are chiefly perceived by the edges of the organ. *Saline* tastes are tested with solutions of common salt, bromide or iodide of potassium, bicarbonate of soda, etc. In addition, cakes, sauces, etc., may be occasionally employed as tests. The testing of the minimum amounts of concentration required to produce gustatory sensations, as well as the plan of testing the sensibility for different degrees of concentration of soluble sapid substances, has not as yet proved of any practical value.

We possess in the *galvanic method of testing the sense of taste* a very valuable and convenient means of investigation for pathological cases. Neumann was the first to show how it may be used with precision. Two fine wires, provided with small knobbed ends, and carefully isolated from one another by means of sealing-wax, are to be attached, at a distance of a few millimetres from each other, to a non-conducting handle, such as a glass rod or an elastic catheter; these wires, which form the electrodes, are then connected with the poles of one or several galvanic elements. If these are placed upon the tongue, a slight burning sensation is felt with a distinct sensation of taste, which is described as being sourish, saline, metallic, coppery, etc., and which is somewhat stronger at the anode than at the cathode. By this means a very exact localization of the galvanic gustatory stimulus is obtained, and it is possible to determine to a hair's breadth, by moving the small pair of electrodes over the surface of the tongue and palate, the limits of the gustatory and non-gustatory area, as well as to form an accurate conclusion in regard to the intensity of the sense of taste in symmetrically situated parts. The gustatory nerves are, it is well known, very easily stimulated by the galvanic current. Distinct galvanic gustatory sensations often occur with great distinctness when the throat, head, neck, and even the back are galvanized ; these are probably caused in most instances by induction currents, which reach the structures in the oral cavity, but it is at the same time possible that the gustatory nerves in their peripheral and central course occasion gustatory sensations on galvanic irritation, and thus a conclusion may be drawn in many pathological cases, by varying the locality at which the galvanic stimulus is applied, in regard to the seat of the lesion which occasions the impairment of the sense of taste. It would be expedient to notice this point in future in pathological cases ; the experiments must, however, be performed with great care, if they are to lead to satisfactory results.

Neuroses of the sense of taste may be classed together in two groups, of which one includes the hyperæsthesiæ and paræsthesiæ of the gustatory nerves, and the other the various forms of anæsthesia.

Hyperæsthesia and Paræsthesia of the Gustatory Nerves.—Hyperæsthesia gustatoria.

These are upon the whole of rare occurrence, and up to the present time are of no great practical importance, but still deserve a somewhat more careful investigation. True gustatory hyperæsthesia may express itself as an increase in the delicacy of the gustatory sensation, so that extremely small quantities of sapid substances may be perceived ; as, for instance, in hysterical patients, who frequently distinguish certain ingredients in food or medicine which are quite imperceptible to a healthy person ; or it may express itself by an increased enjoyment or loathing of food, so that many substances convey a more disgusting or a more agreeable taste than they do in the case of a healthy palate. These anomalies occur chiefly in hysterical patients. Wernich observed a somewhat similar condition in a patient who, after subcutaneous injections of morphia, experienced in a very short space of time, a disgustingly bitter taste on the tongue.

Amongst the *paræsthesiæ* of the sense of taste, we may include the spontaneous subjective sensations of taste which are perceived in the anterior half of the tongue in many cases of facial paralysis of rheumatic origin (Roux, Stich); these are sometimes described as sourish, sometimes as sweetish or insipid in taste. In disease of the fifth pair of nerves, in facial and lingual neuralgiæ, these disturbances of the gustatory sense have not been noticed. To this category belong also the gustatory sensations which are perceived in the tongue when certain drugs have been taken; best known of these is the bitter taste after the use of santonine (Rose), which is so strongly marked that water has a bitterish taste when swallowed. In these cases there is probably some action on the peripheral apparatus of taste. The subjective gustatory sensations of insane patients, partly consisting of hallucinations of taste and partly of illusions, usually disagreeable in character, are unquestionably of centric origin.

Anæsthesia of the Gustatory Nerves.—Anæsthesia Gustatoria.[1] Under this name are included the various tolerably frequent forms and degrees of impairment of the sense of taste, embracing even complete loss or paralysis of the sense of taste. This anæsthesia may, as regards its intensity, be either complete or incomplete, and, like the cutaneous anæsthesiæ, it may be either total or partial; that is to say, the loss of the power of taste may include all the varieties of sapid qualities, or only a few of them. It is difficult to establish with precision cases of partial paralysis of taste, since sensibility for different kinds of flavor varies to some extent, even under normal conditions, in various parts of the tongue. Jacubowitch[2] has described a case of partial paralysis of the sense of taste in a leprous Cossack. The patient was unable to perceive either bitter or acid flavors, but, on the other hand, recognized perfectly sweets and salines. No further details are given. Lastly, the paralysis of the gustatory sense may be circumscribed or diffused, in regard to its anatomical distribution, affecting either the tip of the tongue, its root, or one or both sides.

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[1] The term *ageustia*, which is frequently employed to indicate this condition, is decidedly erroneous; it really signifies "the fasting condition," or "having nothing to eat." The term *ageusis* (from γεῦσις=gustatory sense) is alone correct, though in its place the term *ageusia* may perhaps be used.

[2] See Hofmann and Schwalbe, in the Jahresbericht für Anatomie und Physiologie, 1872.

In the next place, *peripheric* gustatory anæsthesia may be produced by all conditions which prevent or render difficult the action of sapid substances upon the gustatory nerves, or which lower the excitability of the peripheral terminal apparatus. Diseases of the lingual mucous membrane, great dryness or a thick fur, act in the first method, the influence of cold or heat, in the latter; it is well known that ice taken into the mouth interferes with the delicacy of the sense, and that very hot foods are not recognized with precision.

The anæsthesiæ of the conducting fibres of the gustatory nerves are much more important. From what has been said above, the anæsthesia may occur in the course of the glosso-pharyngeal, trigeminal, and lingual nerves, and also in the course of the chorda tympani and facial, and by this means important diagnostic points for the determination of the seat of the disease in these nerves is obtained.

In cases of inhibition of conduction in the *glosso-pharyngeal nerve*, taste is abolished in the corresponding side of the root of the tongue, palate, and pharynx. No pathological case has as yet been recorded in which isolated disease of the glosso-pharyngeal nerve has been demonstrated with corresponding and coincident impairment of the sense of taste. The few cases known were for the most part of a very complicated nature, and were accompanied by various lesions of other nerves. We owe to experiments on animals all that is certainly known in regard to paralysis of the glosso-pharyngeal nerve.

In arrest of conduction in the *trigeminus and lingualis*, or in the *chorda tympani* and certain sections of the *facial*, the anæsthesia of the gustatory sense affects the anterior half of the tongue, its borders, and apex; such cases are of very common occurrence, and the seat of the paralysis may be at very different points, and the causes of the paralysis extremely various. In reference to diseases of the *trigeminus* we have already cited a series of cases in which intracranial lesion of this nerve occasioned paralysis of the sense of taste; the number of these cases might easily be increased. So, too, there are several well-established cases in which section of the lingual nerve, performed for the relief of neuralgia, caused complete paralysis of the sense of taste on the opposite side of the tongue (Busch, Inzani, von Graefe, and Vanzetti). Diseases of isolated branches of the trigeminus have been, up to the present time, but rarely proved to be causes of gustatory paralysis; one observation made by Romberg (loc. cit., p. 302, case 3), in which the third branch of the trigeminus was alone diseased, seems to be the only one that is satisfactory in this point of view. The statement of Woodbury, that the sense of taste is lost after section of the third branch of the fifth just below the foramen ovale, is destitute of value, because, in the particular method of operation described, the chorda tympani must certainly have been divided.

Injuries of the *chorda* (inflicted through the external auditory canal), its destruction in consequence of disease of the middle ear and caries of the temporal bone, have been frequently observed as causes producing paralysis of the sense of taste in the anterior half of the tongue. In cases where such lesions are present it is expedient to test the sense of taste from time to time, since the patient does not in many cases perceive spontaneously its impairment.

Paralysis of the *facial nerve* has been frequently observed to be accompanied by disturbance of the sense of taste, however it may have been produced, whether from injuries, from rheumatism, or from affections of the middle ear, or by caries of the temporal bone (Neumann, Roux, Lussana, Tillmanns, Erb; Stich even maintains that he has observed impairment of the taste in all cases [in which this point has been examined] of complete rheumatic paralysis of the facial nerve). In almost all these cases it may be admitted that the lesion of the facial nerve is within the canalis Fallopii and below the ganglion geniculatum.

On the other hand, two cases have been reported, one by Stich, in which the facial nerve was divided immediately below the stylomastoid foramen, and one by Lotzbeck, in which this nerve (or its branches) was divided in front of the ear, in which impairment of the sense of taste, though only amounting to retardation of the gustatory sense, was observed. These cases may be explained on the theory that these are recurrent branches of the chorda, which run through the auriculo-temporal nerve to the brain. It is desirable that it should be deter-

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mined by exact observations whether this impairment of the gustatory sense is a constant phenomenon after section of the facial nerve, as we have found reason, from our own observations, to doubt it. A case bearing on this point is recorded by Vizioli (cited by Lussana in 1872), in which the facial was divided by a stab with a knife behind and below the ear, and which was accompanied by paralysis of the sense of taste, although the author is of opinion that the chorda tympani was also injured.

The influence of paralysis of the facial, caused by intracranial lesion, upon the sense of taste has been but little investigated. I have observed a case in which distinct peripheral paralysis of the facial was coincident with paralysis of the velum palati, and in which, consequently, one would be led to localize the lesion in some part above the ganglion geniculatum. There was here no disturbance of the sense of taste, which is in favor of Schiff's view. Moreover, Stich has never observed impairment of the gustatory sense in numerous cases of intracranial paralysis of the facial.

It thus appears that the anæsthesiæ of the fibres of the gustatory nerves may vary greatly in their nature, and that the correct determination of their origin and seat is often by no means easy. These diffiulties are still further increased by the variations which probably often occur in the course of the gustatory nerves. And inasmuch as these pursue different paths, which may contain the gustatory nerves in different relative proportions, it is intelligible how cases that are apparently quite similar to one another, occurring in different individuals, may present considerable differences in the degree and character of the impairment of the sense of taste.

Before the impairment of the sense of taste can be estimated at its true value as a means of diagnosis for the various paralyses of the nerves above mentioned, a great number of exact observations made with regard to the point in question must be undertaken; our knowledge of gustatory paralyses will only by this means become accurate, and it will gradually be determined whether the above representation of the course of the gustatory nerves is correct, or whether it must be modified. In the meanwhile, what is already known must form the basis for further investigations. The questions before us can only be answered by pathological observations, and the material at present in our possession is far too scanty for any accurate conclusions to be deduced.

Our knowledge is very limited in regard to anæsthesia of the conducting fibres of the gustatory nerves in their centric course; the paths pursued by these fibres are for the most part unknown. We may consider central anæsthesia of the conducting fibres to be present if the areas of distribution of the lingual and glosso-pharyngeal nerves are coincidentally rendered anæsthetic; and if there be, in addition, extensive cutaneous and muscular anæsthesiæ (see Guttmann, Berl. Klin. Woch., 1869; Binz, Deutsch. Klin., 1858). Such cases are most frequently observed in hysteria. In the relatively rare cases of tabes, bulbar paralysis, apoplexy, etc., in which disturbances of the sense of taste are exhibited, this occurs probably also from lesion of the central gustatory fibres. The case of Steiner, which presented disturbance of the gustatory nerve with centric paralysis of the facial on both sides, is not satisfactory, because, on the one hand, it was not certainly proved that the paralysis of the facial was really of centric origin, nor, on the other hand, that the trigeminus was free from disease. The patient experienced frequent and severe burning pain in the borders and tip of the tongue.

We know little or nothing in regard to the paralysis of the sense of taste resulting from lesion of the perceptive centre of the sense of taste.

The *diagnosis* of the degree and of the distribution of the gustatory anæsthesia can only be based upon objective investigation, since no reliance can be placed upon the subjective sensations and the statements of the patient on this point. The diagnosis of the true seat of the disturbance is frequently, as may be deduced from the foregoing statement, beset with great difficulties, and all that has been said in regard to the necessity of exact observation of the concomitant symptoms and of coincident affections of other nerves, is applicable here. It may be mentioned once more that we must be prepared for the possibility of the existence of numerous irregularities in the course of the gustatory nerve fibres. But this should only be a spur to more exact observation. Moreover, the possibility of partial interchange of the fibres in the communications between the glosso-pharyngeus and lingualis must be borne in mind.

The *prognosis* of gustatory anæsthesia is founded upon the nature of the primary lesion; and since the subjective disturbances are proportionally small, the patients in most instances attribute little importance to the existence of this symptom. But it may be remarked that the paralysis of the sense of taste is often extremely obstinate, and not unfrequently long outlasts the paralysis of the facial, through which nerve the gustatory nerve fibres reach the brain.

The *treatment* must, in the greater number of the cases, be directed to the causal indications, and to the removal of the primary lesion; upon which point we must refer the reader to the several sections of this work that bear upon these matters. As a direct means of treating the gustatory paralysis nothing is superior to faradisation or galvanization applied, as may be most appropriate, to the seat and nature of the lesion, either peripherically to the tongue itself, or through the lingualis, the temporal region, etc.

2. Neuroses of the Olfactory Nerves.

Romberg, Nervenkrankheiten, I. 3. Aufl. pp. 157 und 298. 1853.— *A. Eulenburg*, l. c., p. 307. — *Lockemann*, Zur Casuistik der Geruchsanomalien. Zeitschr. f. rat. Med. 3. Reihe. XII. 1861. —*W. Stricker*, Verlust des Geruchs in Folge local. Anästhesie. Virch. Arch. Band 41. 1868.— *Prévost*, Atrophie des nerfs olfactifs fréquente chez les vieillards, etc. Gaz. méd. de Paris. 1866, No. 37. — *Jackson*, Remarks on the Occurrence of Hallucinations of the Sense of Smell in Epilepsy, Lancet, 1866. Jan. 24. — *Notta*, Recherches sur la perte de l'odorat. Arch. génér. de Méd. Avril 1870 (Reports of numerous cases). —*W. Ogle*, Anosmia, or cases illustrat. the physiol. and pathol. of the sense of smell. Med.-chir. Transact. LIII. 1870 (valuable treatise). — *Mollière*, Note pour servir à l'histoire de la pathol. du nèrf olfactif. Lyon méd. 1871, No. 20. — *Sander*, Epil. Anfälle mit subject. Geruchsempfindung bei Zerstör. des l. Tract. olf. durch einen Tumor. Arch. f. Psych, und Nerv. IV. p. 234. 1873. — *Althaus*. Treatise on medical electric. II. ed. 1870. — *Beard* and *Rockwell*, Medical and surgical electricity. 1870.

The anatomical and physiological relations of the olfactory apparatus are comparatively simple, at least so far as pathology is concerned. The olfactory nerve is the only nerve of smell. Its branches, after traversing the lamina cribrosa of the ethmoid, are distributed to a limited portion of the Schneiderian membrane (the upper part of the septum, and upper and middle turbinated bones), which is termed the regio olfactoria. The terminal branches of this nerve are probably in immediate connection with certain peculiar terminal organs which receive olfactory impressions and conduct them to the nerve fibres. Nothing certain, however, is known in regard to the central course of the olfactory nerves. The so-called external root of the tractus olfactorius contains the greater number of fibres, and may be followed centrally into the neighborhood of the island of Reil.

The first and second divisions of the fifth constitute the true sensory nerves of the mucous membrane of the nose, but these have nothing to do with the perception of odors; they conduct only tactile and common sensations.

The specific sensations of smell are caused by a countless number of volatile gaseous bodies, each of which produces a definite sensation which is not comparable with any other. The precise nature of sensations of smell cannot be defined more exactly than those of taste. In them also the concomitant feeling of pleasure or dislike, of enjoyment or loathing, is usually very well marked.

The olfactory nerves respond very feebly to electric stimuli. According to Althaus, if the nasal mucous membrane be irritated with very strong galvanic currents, a taste as of phosphorus is perceived. No perception of odors, however, is, for the most part, perceived, notwithstanding the occurrence of coincident strongly marked secondary phenomena, such as pain, vertigo, and sensations of light. On this account the galvanic current cannot be employed for testing the sense of smell.

The testing of this sense can be very much better accomplished by making the patient smell different odorous substances with the two nostrils alternately, avoiding, of course, all materials which act as irritants upon the fibres of the fifth, as, for example, ammonia, acetic acid, and snuff, for all these cause tickling or pricking sensations and sneezing, and thus disturb considerably the true sensation of smell. It is best to select odorous materials which are partly pleasant and partly unpleasant. Amongst the first group, that is to say of agreeably smelling bodies, flowers, eau de cologne, oil of bergamot, oil of rosemary, musk, camphor, and the like, may be mentioned; whilst amongst the other group of disagreeably smelling bodies are valerian, assafœtida, turpentine, sulphuretted hydrogen, and other bodies well known in organic chemistry. In order to complete the testing, it is expedient to test the perception of the flavor of aromatic or agreeably tasting fluids and solids, as, for example, vanilla, cinnamon, cheese, coffee, wine and liqueurs, because the special flavor of each of these depends, in great part, on the participation of the sense of smell, and it frequently occurs that the sense of smell may thus be called into action, while it is impossible to do so through the nasal openings (see the pathological cases reported below).

The neuroses of the olfactory nerve, in regard to the pathology of which little is at present known, may be grouped in very much the same manner as the neuroses of the gustatory nerves.

Hyperæsthesia and Paræsthesia of the Olfactory Nerves.—Hyperæsthesia Olfactoria.

True simple hyperæsthesia of the sense of smell—diminution of the perceptible minimum-stimulus—occurs now and then in hysterical patients, who often perceive, and can distinguish, almost inconceivably minute traces of odorous substances, materials, and persons.

As occurs also in the sense of taste, hyperæsthesia of the sense of smell may also be associated with great exaltation of the concomitant feelings of pleasure or disgust, the latter being often extraordinarily well marked. Odors scarcely perceptible, or which are indifferent, or even pleasant perfumes, the odor of flowers, etc., excite in many patients the most disgusting, disagreeable, and even painful sensations, occasioning headache, fainting, and convulsive attacks. On the other hand, it may happen that nauseous and unpleasant odors are tolerated by the patient, and may even be preferred. The greater number of these cases are also hysterical, but the same symptoms are frequently observed in insane patients.

Subjective sensations of smell of the most various kinds occur in many diseases of the olfactory nerves and of the brain, both in epileptics and in insane patients. Unpleasant and disagreeable sensations of smell are generally complained of, such as the smell of gas, of putrefaction, of the fumes of sulphur, of some "horrible" odor, etc., whilst indifferent or pleasant odors are rarely perceived. Tumors, softening and degeneration of the olfactory nerve are usually accompanied by such subjective sensations of smell, which disappear with the complete destruction of the nerve, giving place to anosmia. These sensations are purely excentric sensations, caused by irritation of some part of the olfactory nerves. Such paræsthesiæ of the sense of smell occur in the most various forms of cerebral disease (Maingault, Dubois, Lockemann, Westphal, Sander, Schlager). In insane patients, such subjective sensations of the sense of smell are not unfrequent; they are either true hallucinations or more frequently illusions, and are for the most part unpleasant or highly disgusting; they are also frequently the source of various insane ideas and abnormal actions. Lastly, subjective olfactory sensations of this kind have been frequently observed in epileptics, partly occurring in an independent manner between the attacks, and partly and more frequently preceding them as a kind of aura (Jackson, Lockemann, and Sander).

Up to the present time nothing is known in regard to the proper *treatment* of these hyperæsthesiæ. The principal point to be attended to is the treatment of the primary disease. In many cases it would be right to try the effects of anæsthetics or of electricity.

Anæsthesia of the Olfactory Nerves.—Anæsthesia Olfactoria. Anosmia.

This disease is both more frequent and more common than hyperæsthesia. Under the term anosmia, however, many conditions have been grouped, which have scarcely anything to do with the nervous apparatus of smell. Thus it has been customary to include under this head all conditions in which the sense of smell is lost, even when the olfactory region, with its sensory nervous apparatus, is quite healthy. On this account we are compelled in the following observations, for the sake of completeness, to mention many things which do not strictly belong to this affection, but still require to be briefly mentioned. The reader will easily make the necessary distinction.

Anosmia expresses itself by progressive diminution in the intensity of the perception of smell, which may proceed to its complete extinction. In the first instance it often occurs that only a few special odors fail to be perceived, especially the delicate perfumes, etc., whilst others are often long preserved. Ultimately, however, the sense of smell is lost for all kinds of odors. The sensibility of the Schneiderian membrane, which is supplied by the fifth nerve, is however perfectly preserved, the stinging sensation of ammonia and of acetic acid being as distinctly perceived as before, and tobacco occasioning sneezing, even though its peculiar smell is not perceived.

In almost all instances the patients complain of a very considerable impairment of the sense of taste, though it is only in cases where the anosmia affects both sides. They are unable any longer to distinguish with accuracy the various kinds of food and wine, and have no perception of different flavors. If their gustatory sense be more closely examined, it soon becomes evident that they are quite able to perceive the four principal kinds of sapid substances, sweet, acid, bitter, and saline ; on the other hand, all those impressions produced by the various kinds of food, and which are termed by the Germans "*Wohlgeschmack*," by the French "*Saveur*," and by the English "Flavor," are lost to such patients. Coffee gives the impression of sweetened hot water with a slight bitterish taste ; wine, of weak vinegar, with more or less roughness ; cheese, as something saline ; and, in short, the different kinds of food have lost their aroma and distinctive flavor. Cases, however, do occur in which the perception of flavor is preserved, even when the anosmia affects both sides. Ogle has explained these phenomena in a very satisfactory manner.

Taste is limited to the well-known four chief categories mentioned above, or to a few allied simple perceptions; all other sensations received from food and drink—the simple tactile impressions, which enable us to draw conclusions in regard to the consistence, smoothness, roughness, tenacity, condition of friability, etc., of the ingesta being excepted—belong to the sense of smell; it is the special means of conducting the so-called sensations of flavor, which we know and enjoy under the names of the "bouquet" of wine, the "aroma" of roast-meat, and the delicacy of aromatic substances; it confers upon articles of diet their chief seasoning. We are accustomed in taking food to rely upon the co-operation of both senses, that of taste and that of smell, including both under the term of taste. Pathological cases, however, enable us to distinguish these two sensory perceptions; thus, if smell be lost and taste preserved, flavors are no longer perceived, and only the simple perceptions of taste remain. This explains the remarkable alteration noticed under the circumstances in the taste of most of our articles of food and drink.

Since the sensations communicated by the sense of smell are more important and more characteristic, the disturbance of the taste, when the sense of smell is lost, appears much more marked to the patient than in cases of gustatory paralysis. How often true paralyses of the sense of taste are completely lost sight of by the patient! And then, on the other hand, complete gustatory paralyses are of rare occurrence. The disturbance of the taste in bilateral anosmia thus admits of ready explanation.

But the apparently paradoxical fact, that, with complete inability to perceive odorous substances inhaled through the nose, the flavor—that is to say, the sensation of smell proceeding from food and drink—may still be preserved, may likewise, when carefully considered, be explained with equal facility. True odors are communicated by streams of air passing from the nostrils to the olfactory region; flavors, by odorous materials passing from the pharynx through the choanæ to the olfactory region. Now, the nasal cavities, in consequence of the close approximation of the middle turbinated bone to the septum, are divided into two canals, of which the upper and anterior is chiefly subservient to the olfactory current, the lower and posterior to the respiratory current of air. The entrance of air into both canals is facilitated by certain muscular movements which expand the nostrils, whilst by other muscular movements, on the contrary, as by the action of the compressor narium, the respiratory canal is almost completely occluded, and the current directed almost entirely through the olfactory canal, as in the so-called action of sniffing (Ogle). It is consequently necessary for smell that the entrance to the olfactory canal should be sufficiently patent. If the requisite muscular movements be defective, as occurs in facial paralyses, or if the anterior entrance of the olfactory canal be closed by swelling of the mucous membrane, by polypi or other conditions, no current of air penetrates into the olfactory canal even in free respiration, and no trace of the sensation of smell is perceived. Hence the possibility that air charged with odorous substances may enter the olfactory region from the choanæ, when no obstacles of this kind exist, and be clearly perceived, and the sensations of flavor for the various kinds of food and drink, may thus be conveyed. In this way those cases may doubtless be explained in which, though the proper sense of smell is lost, the flavor of food and drink is preserved. For examples the reader is referred to Notta and Ogle. In all such cases the conclusion is admissible, that the capacity for smell is preserved, but that the access of odorous substances to the olfactory region is rendered impossible through the nostrils, but is free through the choanæ. An interesting case in point has been communicated by Ogle, in which the nasal cavities were shut off from the oral cavity by adhesion of the soft palate to the posterior pharyngeal wall ; as a consequence of which both the sensations of smell and of flavor, with the exception of the simple gustatory sensations, were lost, though as soon as the communications were established, both were again perceived.

Anosmia may originate in very different modes : in the first place, peripherically, by all circumstances that obstruct or render impossible the action of odorous substances upon the olfactory apparatus ; and, secondly, by all circumstances that lower or abolish the excitability and receptivity of this apparatus. The latter cases alone constitute examples of true anosmia. The two categories, however, are not always easily distinguishable.

One of the commonest causes of anosmia is catarrh of the nasal mucous membrane. Acute and chronic coryza, influenza, etc., may abolish the sense of smell for a variable period, or even permanently ; this occurs from direct trophic disturbances of the terminal apparatus of the nerves of smell, but in part also from mechanical constriction or occlusion of the olfactory canal. Abnormal dryness of the mucous membrane of the nose is also a cause of anosmia. It is in fact the intermediate condition leading to anosmia of the same side in many cases of anæsthesia of the trigeminus or paralysis of the facial. In anæsthesia of the trigeminus, the secretion of the mucous membrane of the nose is diminished, or the lachrymal secretion ceases, and consequently the moisture of the Schneiderian membrane requisite for the sense of smell is interfered with. In paralyses of the facial the discharge of the tears into the nasal cavity is rendered impossible by imperfect closure of the lids, and in this way anosmia results. But the coincidently present paralysis of the levator alæ nasi and of the compressor naris constitutes an additional favoring circumstance of great importance. Constrictions of the nostrils, nasal polypi, occlusion of the nasal and pharyngeal cavities, all act in a similar mechanical method, that is to say, by simple prevention of the circulation of air through the olfactory canal. Loss of smell has been observed (by von Franque) after catching a severe cold, but no explanation has hitherto been given of this phenomenon. Amongst other peripheral causes of anosmia, which have been observed, may be enumerated strongly stimulating smells (Romberg), stimulating injections and nasal douches, ammonia, sewer gas (Graves), and ether (Stricker).

Lastly, Ogle has referred to the importance of the pigment of the olfactory region for the sense of smell, and has endeavored to establish the fact in a very interesting manner. He sought to explain a case of anosmia which occurred in a negro boy, coincidently with general loss of pigment in the skin (reported by Dr. Hutchinson), by attributing the loss of smell to the disappearance of the pigment in the olfactory mucous membrane.

The forms of anosmia which originate in *inhibition of conduction in the olfactory nerves* and in the tractus olfactorius, are much more important than the above-mentioned peripheral anosmiæ; they are, for the most part, accompanied by subjective sensations of smell. To this class belong, for the most part, traumatic anosmiæ, which result from falls on the head and especially on the occiput, but are not always accompanied by fractures of the skull (Mollière, Notta, Ogle). The olfactory nerves are of course generally affected in direct fractures of the frontal and ethmoid bones; but Ogle also admits that a detachment of the bulbus olfactorius from its branches traversing the lamina cribrosa may occur without fracture of the skull, and anosmia may be the consequence.

Prévost has demonstrated degeneration and atrophy of the olfactory nerves as causes of senile anosmia, which is not unfrequent in elderly persons. So, too, softening and induration of the nerve, or the formation of a tumor in it, tumors in the anterior fossa of the skull and in one of .the anterior lobes of the brain, exostoses, caries, and meningitis near the part in question, have been frequently shown to be causes of anosmia (Loder, Oppert, Romberg, and others). To all these we must add the cases of congenital anosmia (Notta), in explanation of which several observations, as those by Cerutti, Rosenmueller, and Pressat, have demonstrated the absence of the tractus olfactorius.

Many cases of anosmia are of centric origin, and are due to arrest of intracranial conduction, or to inexcitability of the olfactory centre. The cases of anosmia which accompany aphasia and dextral hemiplegia, and are always limited to the left nasal cavity, are particularly interesting (Jackson; Fletcher and Ransome; Ogle; I have also myself a case at the present time under treatment, caused by embolism). Ogle calls attention to the fact that in these cases there is concomitant disease of the external root of the left olfactory tract, which may be followed as far as the island of Reil, and that this fact may hereafter possess a certain diagnostic value in the localization of cerebral diseases. The anosmia that occurs so frequently in hysteria is also of central origin; it is for the most part associated with loss of taste, with cutaneous and muscular anæsthesiæ. The anosmia that occurs in the insane is also of central origin.

It is impossible to state what is the seat of the disease producing anosmia in some of the cases mentioned by Notta, who applies to them the term "essential anosmia;" the loss of smell appeared without any apparent cause in young women, and was unaccompanied by any other disturbance.

The *prognosis* of anosmia depends chiefly upon the cause. Those cases which result from serious organic lesions are incurable. A more favorable prognosis may be given in anosmia proceeding from coryza, exposure to cold, traumatism, etc., and those are most favorable which depend upon mechanical conditions, provided the obstruction to the entrance of a current of air into the olfactory canal can be removed. Since the loss of the sense of smell involves a loss of a larger number of unpleasant than of pleasant odors, patients usually attach but little importance to the absence of this sense. The complete loss of the sense of flavor, which accompanies it, is alone complained of as being very unpleasant.

The *treatment* has up to the present time proved very unsatisfactory. For the most part it must be limited to the treatment of the primary disease and to the removal of such causes as may be ascertained to be present. Direct treatment has not hitherto proved very successful. Notta saw only negative results follow the employment of various irritating snuffs, but observed that many chronic cases recover without any treatment at all. Duchenne has observed good results to follow faradisation of the nasal mucous membrane, especially in hysterical patients. Beard and Rockwell also recommend faradisation and galvanisation. These agents they apply partly outside the nose, partly by means of a sound-like electrode, directly to the mucous membrane. Fieber (Electrotherapie) saw good results follow from the application of the galvanic current by means of olive-shaped electrodes in both nostrils.

NEUROSES OF THE MOTOR NERVES.

The essence of *neuroses of motility* consists in disturbance of the function of those centrifugal nerve paths which are recognized as motor, and of the central and peripheral terminal apparatus connected with them. The relations are far more complicated in the neuroses of motility than in those of sensibility, because the excitation of the motor functions may proceed from more numerous points.

A glance at the normal anatomical and physiological relations of the motor apparatus teaches in what way and from what causes pathological disturbances of motility may arise.

The so-called motor nerves, with their peripheral terminal apparatus, the muscles, form the anatomical basis of the motor processes. The muscles constitute essential parts of the apparatus of active movement, since without them the motor activity of the nerves, a motor function, would either be impossible or at least not perceptible to us. The view therefore is not unjustifiable, that they should be regarded as constituting, with the motor nerves, one whole and indivisible organ, and that disturbances, which proceed from changes of the muscles, should likewise be included amongst the motor neuroses. Strictly speaking, however, the conception of motor neuroses must be limited to disturbances of the motor nerves, including the terminal apparatus (motor terminal plates, etc.) in the interior of the muscular fibres. It is obvious that so strict a limitation as this is often impossible in practice, changes taking place in the intra-muscular endings of the nerves, and in the muscular fibres themselves which are often indistinguishable from one another. This fact must always be borne in mind in describing the different neuroses, though perhaps such a confounding of the different processes may not stand the test of strict criticism.

On following the motor nerves to the spinal cord, we find that they enter it by the anterior roots, but their subsequent course has not as yet been satisfactorily ascertained; they probably pass for the most part from the anterior roots into the gray substance, and after remaining for a short distance in this, they re-enter the white medullary antero-lateral columns, in which they continue to the level of the medulla oblongata, decussating only to a slight extent in the spinal cord. In addition to this principal course, there are, however, numerous secondary paths, which probably all lie in the gray "kinesodic" substance, and serve for the conduction of motor impulses under certain physiological and pathological conditions.

The principal decussation of the motor nerves takes place at the level of the medulla oblongata and pons; beyond these structures—in the crus cerebri and still higher—only motor fibres that have decussated are to be found. This is true also of all those motor fibres in the cerebral motor nerves, which pass directly to the brain.

It is only necessary to refer here very briefly to the course of the motor fibres in the brain. The larger number runs from the pons through the pes of the crus cerebri to the great central ganglia (corpus striatum and lenticular nucleus) with which some of the fibres form important communications; beyond this, the fibres are continuous with those of the corona radiata, and run to the gray substance of the hemispheres, terminating chiefly in the anterior lobes. The fibres here probably end in certain motor centres, the existence of which has been demonstrated by Fritsch and Hitzig. The various groups of muscles, as those of the face, the eyes, the upper and lower extremities, trunk, etc., possess separate centres, and by electrical excitation applied to these centres, movement can be excited in the corresponding groups of muscles. Whether these motor centres also represent the centres of voluntary activity, is still undetermined, but it is certain that the transference of voluntary impulses to the muscles is dependent on the integrity of these centres.

In addition to this principal course pursued by the motor nerves, there are many communications, secondary paths, etc., by which excitation may be conducted through motor fibres. Thus, in the first place, a communication exists in the spinal cord between motor and sensory fibres, by means of which reflex movements are effected, a connection which is undoubtedly accomplished by means of ganglionic cells and occurs in the gray matter; similar connections are found also at higher planes, in the medulla oblongata, and in the brain; these are uncommonly numerous and widespread, and are of great importance. Many connections of the motor nerves with so-called automatic centres, from which direct excitations proceed, occur in the medulla oblongata and brain, of which the centres for the respiratory and cardiac movements, and for the vaso-motor paths, may be cited as examples. Still more complex and still less carefully investigated are, lastly, the relations of the motor nerves to the various parts of the cerebrum (corpora quadrigemina, optic thalami) and cerebellum, in regard to which we cannot here enter into further details.

The physiological functions of the motor apparatus may be arranged under different heads according to the kind and mode of the excitation which primarily occasions the muscular contraction. They are as follows :

Voluntary movements—expressions of the conscious will, proceeding from excitations of the central apparatus of the will (situated very probably in the gray substance of the cerebral cortex), and transferred to motor fibres.—Closely allied to these are the *associated movements*, which result from the communication of voluntary impulses to motor nerves not directly intended to be called into play. The occurrence of these movements is partly referable to the contiguity of the several motor centres of the various groups of muscles in the cerebral cortex, as in the case of the facial and ocular muscles (Hitzig), partly to numerous secondary connections and paths, which are everywhere found in the gray substance, and which render possible such an extension of an original stimulus to numerous nerves. Associated movements, which frequently occur physiologically, and which also play a certain part in pathology, may occur, not only in voluntary movements, but also in other forms of motor excitation.

Automatic movements.—These are excited by the action of certain centres independently of the will, the excitation itself being ordinarily caused by certain conditions and changes in the blood. The centres for the respiratory and cardiac movements, for vomiting and for the vaso-motor nerves, constitute such automatic centres, and are, for the most part, also capable of being excited in a reflex manner. They are all contained in the medulla oblongata and in the brain. Whether automatic movements are effected through the intervention of the spinal cord has not been certainly ascertained ; the much contested existence of

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muscular tone might be explained as a movement or excitation of this kind, but, since its existence, at least in the voluntary muscles, is doubtful, and since it has no special importance in pathology, the question will not here be discussed.

Reflex movements. These are the consequences of the direct transference of centripetal excitations to centrifugal paths, without the co-operation of the will. The physiological laws governing reflex movements are, we presume, well known, and are often distinctly recognizable in pathological cases. All possible degrees, from the simplest to the most complex movements, occur in pathological conditions. The phenomena of *reflex inhibition*, which often oppose reflex movement, and which usually result from coincident excitation of sensory nerves, must not be overlooked. The chief centres of these inhibitory processes are situated in the brain (constituting the inhibitory reflex centres of Setchenow), and from these a pathological modification of reflex movements may proceed.

Lastly, certain processes may here be briefly referred to, which play a very important part in the manifold forms and disturbances of movement, though as yet very little light has been shed upon them. To this category belong the *processes of combination and co-ordination of movements*, or the employment of definite motor paths for the performance of coincident advantageous action, as is observed, for example, in speaking, writing, walking, playing the piano, etc. These and similar processes frequently come under our consideration, and demand further investigation.

From what has been said above, it is easily intelligible how numerous and complicated the disturbances of motility may be; from how many different points abnormal excitations may be excited in motor paths, and at how many points inhibition of motor impulses may occur. In fact, the forms and kinds of neuroses of motility are extremely numerous and diverse, and the recognition of the primary causes is often very difficult, and, in many cases, even impossible.

We must here limit our remarks to those disturbances which originate in disease of the peripheric motor nerves, or their virtual prolongations in the spinal cord and brain, so far at least as they fall under the vague denomination of the so-called neuroses. Those disturbances will now be chiefly discussed which occur in the paths of voluntary movements, or in reflex paths, or in the paths of certain automatic movements. The more complicated disturbances which proceed from the central apparatus connected with the motor nerves will be discussed to better advantage when the diseases of the central organs are under consideration.

Disturbances of motility (of the motor processes chiefly) may be of two kinds:

Either an exaltation of the motor phenomena is observed, which may differ in kind and degree, and which is termed *hyperkinesis*, or *spasm*; or

A diminution of the motor phenomena is observed which may proceed to their complete extinction, and which is termed *akinesis* or *paralysis*.

The changes, therefore, which we encounter under pathological conditions are, for the most part, *quantitative* changes of motor activity. It is doubtful whether *qualitative* changes can occur. The various forms of convulsion may indeed be regarded as such, though, strictly speaking, not correctly so; for in these we have only to deal with the differences that result from variations in the order of succession, the extent, and the intensity of motor excitations. While the causes which are at work in producing the excitations may differ in quality, the result produced can only be a quantitatively modified excitation (muscular contraction).

Mode of examining motility and motor apparatus.—A few practical observations may here be made in regard to the mode of making an examination into the nature of neuroses of motility. Our remarks, however, will be limited to those points that are practically useful and important.

The functional activity of the motor apparatus expresses itself exclusively by contraction of the muscles, and by the movements of the body, or of particular parts of it, which are thus occasioned. In order, therefore, to test the physiological action and the pathological disturbances of the motor apparatus, it is obvious that the observation of muscular movements, that is to say, the testing of the position and movements of the whole body, or of its several parts, is of the utmost importance. This is, of course, best accomplished in the naked body, though many of the more marked anomalies can be easily perceived when the patient is dressed.

A practised eye immediately recognizes certain forms of anomaly; and it is indeed in many cases easy to make the diagnosis of various motor disturbances at the first glance. A very minute and exact investigation, however, is often requisite, in order to render the characteristics of the affection evident. These disturbances are recognizable partly in consequence of certain positions and movements (as in cramp) being too strongly marked, and partly by the feebleness with which certain movements are performed; or by their entire absence, and the consequent anomalies which are produced in the general position, configuration and symmetry of the body, or particular parts of the body (as in paralysis).

An exact knowledge of the physiological function of the different muscles is absolutely requisite for the recognition of the first group of disturbances. With this foundation to build upon, the diagnosis is for the most part easy; in complicated cases, however, and where there are cramps of particular and deeply situated muscles, which ordinarily act co-ordinately with others, the difficulties are very great, and require the exercise of extreme care to overcome them. The chief aids to this investigation are an examination made as completely as possible of the whole naked body, the performance of active and passive antagonistic movements, the interposition of some passive resistance to the cramp-like movements, and the forcible extension of the abnormally placed parts of the body.

An excellent preparatory means of investigating these conditions consists in the local faradisation of such muscles as may be accessible. By this means an artificial spasm can be produced in the muscles in question, the result of which is, so far as the purposes of observation are concerned, identical with that furnished by spontaneous cramps. Local faradisation of antagonistic muscles, and of the symmetrical muscles of the other half of the body, may also be used to aid the diagnosis.

Lastly, certain voluntary movements (on the part of the patient) may be made by an effort of the will, in order that the disturbances may be recognized by the changes which are often first observable when such voluntary movements are attempted. In this way complicated movements must also be examined, as those of standing, walking, hopping, writing, piano-playing, sewing, and the like.

For the exact recognition of the inability to perform certain movements it is necessary to investigate many special points.

The question first arises, *what movements* are enfeebled or abolished, and we have thus, in the first instance, to inquire into the preservation or absence of the movements of particular muscles or groups of muscles. For this purpose the several voluntary movements must be isolated; hence physiological knowledge is absolutely indispensable to the physician in order that he may be able to make the patient perform the necessary movements. The acquirement of this knowledge has been materially facilitated by the excellent works of Duchenne[1] and of Ziemssen.[2] In regard to the face, it is necessary to investigate the voluntary movements and those mimetic ones that occur in speaking, laughing, etc.

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[1] Physiologie des mouvements, 1867. [2] Electricität in der Medicin, 4te Auflage, 1872.

In the trunk and neck attention should be paid to the actions of flexion and extension, to those of lateral motion and rotation, to the movements of respiration and of abdominal pressure, etc.

In the upper extremities we should observe the actions of flexion, extension, and rotation of the several joints, pronation and supination, the movements of particular fingers, etc.; in the lower extremities, flexion, extension, adduction, abduction, and rotation in the several joints, standing, walking, standing on the toes, standing on one foot, etc. We shall enter into the details of these inquiries when treating of the several forms of paralysis.

In the next place, it is requisite to investigate to *what degree* particular movements have undergone impairment, whether there is a greater or less degree of weakness of voluntary action (*paresis*), or whether these movements are completely abolished (*paralysis*). This can, in most cases, easily be ascertained, but may present great difficulties in the case of the special muscles that are brought into play in complicated movements performed by the co-operation of several muscles.

If the paralysis be not complete, the further question arises, what amount of force can still be exerted by the affected muscles. An approximate estimate can be formed on this point by comparative testing of the resistance which can still be opposed to passive movements, and by testing the pressure which the patient can exert with his extremities. The force capable of being exerted can be measured directly by means of the various forms of the dynamometer, which are partly adapted for pressure and partly for traction; but which can only be applied to a few of the muscles. Such estimates, however, since there is no absolute measure for the amount of force exerted by the several muscles, have only a relative value, and are really serviceable where it is desired to estimate the increase or diminution of force in certain muscles of the same person, or where a comparative investigation has to be made of symmetrical parts of the body. The estimate may be materially assisted by investigating the subjective sensations of the patient, such as the period, whether sooner or later, at which exhaustion is experienced.

Lastly, the degree of uniformity, precision, and certainty with which the several movements are performed must be tested, whether they are accomplished smoothly and evenly or by jerks, and whether they are accompanied with trembling or disturbing secondary movements. With this object in view, the patient must be directed to perform special movements, such as the drawing of straight lines or circles, touching particular points with the fingers, walking along a plank, etc. The deviations that occur in walking may be graphically represented by making the patient walk under a board on which a line is traced by means of a pencil attached to his head. Accurate conclusions may also be drawn from exact observation of various complicated movements, such as those of running, hopping, climbing a chair, writing, sewing and knitting. In respect to many motor disturbances, as cramp, ataxia, paralysis, it is often advantageous to combine with these investigations the testing of the so-called sense of muscular effort (see above).

The *performance of passive movements*, again, enables us to form an opinion in regard to various conditions that may be present; as, for instance, whether the paralyzed muscles are entirely relaxed, or whether they are tense, contracted, etc., whether they are easily thrown into a state of convulsive tremor, what muscles are in a state of cramp, and what is the amount of force exerted by the muscle in its contracted state.

The testing of the *reflex movements* must never be omitted; their preservation, augmentation, or absence often furnishing important diagnostic aid in determining the seat of motor disturbances. The reflex movement of the organs of special sense, as of the eye, ear, and tongue, those of the mucous membranes, as of the conjunctiva, Schneiderian and pharyngeal mucous membranes, etc., and, above all, of the skin, must be tested by tickling, pricking, pinching, faradic excitation, etc. The investigation of the *automatic movements* must also, of course, be undertaken.

The activity of the motor apparatus and their pathological disturbances may, by the use of these means, be rendered tolerably evident. We still, however, possess other means of drawing conclusions in regard to the condition of the motor apparatus (nerves and muscles), even when these have quite lost their physiological activity. These means are to be found in external stimuli, which may be applied to the various parts of the motor apparatus; by these means various reactions, that are often materially modified in pathological conditions, may be called into play. From the anomalies presented by the motor apparatus on stimulation, we are, in many cases, in a position to draw definite conclusions in regard to the anatomical and physiological states of the muscles, and from these states, in turn, some idea may be formed in respect to the seat of the disturbance in the motor apparatus.

Electrical and in part also *mechanical stimuli* are the most appropriate external stimuli for the medical investigation of motor organs; and inasmuch as their application to the purposes of diagnosis supplies us, in many instances, with very valuable indications in respect to many of the more delicate relations in convulsive affections, as well as in paralyses, and will frequently be referred to in the following sections, it will be expedient here to give a short account of the physiological relations that exist in the healthy living human body. This will be done partly to avoid repetition, and partly for the information of those readers who may not be familiar with the special literature of the subject.

The *excitability to mechanical stimuli* of motor nerves and muscles may be tested in the simplest manner by the ordinary percussion hammer. Under normal physiological conditions, a moderately strong blow induces, in most muscles of the body, though more or less easily in different instances, a momentary contraction of the particular muscular fasciculus struck, especially if the blow fall near the point of entrance of the motor nerves. In some muscles, as, for instance, in the pectoralis major, the deltoid, and the extensor muscles of the forearm, these contractions occur with greater facility; in others, with less. In very excitable persons, as in those who are convalescent from serious diseases, phthisical patients, etc., a circumscribed tumor not unfrequently appears at the point struck, from which small waves of contraction run in both directions towards the extremities of the muscles—a form of contraction in regard to which physiology has not thus far given any satisfactory explanation. The nerve trunks themselves are not easily excitable by the direct application of mechanical stimuli, a tolerably strong and often painful blow being requisite in order to produce muscular contraction through them. In such experiments the muscles must be in as relaxed a state as possible. In pathological conditions a remarkable increase of excitability to mechanical stimuli has been observed (Hitzig, Erb).

Electrical excitation of the motor apparatus furnishes us with more important and more widely applicable results. It has already been developed into an extremely valuable means of diagnosis; it enables us to draw conclusions in regard to many palpable as well as impalpable changes in the muscles accompanying the most various motor disturbances; it not unfrequently enables us to judge correctly of the anatomical seat of the lesion; it affords us a basis for the prognosis, and not unfrequently supplies indications for treatment. An accurate insight into the pathology of many motor disturbances could not, at the present time, be obtained without electrical investigation.

It is obvious that an exhaustive account cannot here be given, and we must refer the reader, for information on this subject, to the manuals of physiology and to the more recent works on electro-therapeutics.[1] We shall here limit ourselves to a short account of the effects of electrical excitation applied to the motor nerves and to the muscles of the living healthy man, as far as the facts may be considered to be established by the most recent investigations. We shall have frequent occasion to refer to them in speaking of pathological changes.

The basis of the whole method of examination is the fact that motor nerves and muscles are excited to the performance of their specific functions by the application of electrical currents (whether galvanic or faradic), and that muscular contractions are thus induced; in other words, that motor nerves and muscles react to electrical currents, and that this reaction takes place according to a definite law (law of contraction). The excitation of the motor apparatus results essentially from the variations in the density of the current, that occur coincidently with the closure and opening of the current traversing them. (A certain amount of excitation occurs also during the continuous passage of the current, but is of less importance for our purposes.) The excitation is proportionately stronger the greater these variations of intensity are and the more rapidly they occur, or in other words, the stronger the current and the more sudden the variations of its density; the excitement does not occur when the current is very weak, or where the variations in density are very gradually produced. Upon this fact the whole structure of electrical investigation really rests.

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[1] See, in particular, *Brenner*, Untersuchungen und Beobachtungen auf dem Gebiete der Electrotherapie, Leipzig, 1868-69; *Ziemssen*, Electricität in der Medicin, 4. Auflage, Berlin, 1872; *Erb*, Sammlung Klinischer Vorträge, herausgegeben von Volkmann, No. 46, 1872.

Two kinds of electrical currents are commonly employed for the purposes of electrical research: *faradic* (or induction) *currents*, and *galvanic* (or contact, constant battery) *currents*. The former are generated in automatic machines, which furnish a series of isolated currents, each of momentary duration, and of very rapid development and decline, that follow each other in quick succession, flowing alternately in opposite directions. The latter are continuously produced, and consist of currents running in the same direction and with the same intensity; in these currents, however, we are able, by means of the so-called commutator (Stromwender), to produce interruptions at will, that is to say, the current can be completed and broken, and by these means the necessary variations in density may be caused. For all details on these points the manuals on electro-therapeutics must be consulted.

The behavior of the nerves and muscles under the influence of faradic currents, the kind and strength of the reaction which they exhibit, are comprised under the term *faradic excitability*. It matters not whether the muscular excitation be caused by nerves or by the muscles; one effect alone is produced—muscular contraction. If this is effected by excitation of motor nerves, it is said to be by *indirect excitation;* if by a stimulus applied to the muscles themselves, *direct excitation*. If, therefore, indirect faradic excitation of a muscle be referred to, the excitability of the corresponding motor nerves is implied, whilst by the term, direct faradic excitability, the excitability of the muscular substance is understood. (The same terminology is of course used for galvanic excitation.)

For the purpose of testing the faradic excitability, the "secondary induction currents," obtained from the secondary coil of the ordinary induction apparatus, are usually employed, though the so-called extra current of the primary coil may also be used. The cathode of the opening induction current is usually employed as the exciting pole, and it is therefore expedient, in order to avoid too great complication, to use the cathode exclusively for excitation, and to place the anode upon some indifferent part of the body, as the sternum, patella, etc. Such is the mode of applying electricity to which all our observations refer.

When electricity is thus applied to healthy persons, it is observed that on excitation of all accessible nerves with a feeble current of a given strength (measurable by means of the movable cylinder of the apparatus), a minimum contraction of the muscles takes place, whilst on moderate augmentation of the strength of this current strong tetanic contraction of the muscles occurs; that the symmetrical nerves of the two sides of the body can be excited to minimum contraction by precisely the same strength of current; and, lastly, that various nerves lying immediately beneath the skin of different parts of the body, such as the ramus frontalis nervi facialis, nervus accessorius, nervus ulnaris, and nervus peroneus, can likewise produce minimum contractions when stimulated by currents of about the same intensity.[1] All considerable deviations from these relations must be regarded as pathological, and must be in one or the other direction: either an increase or a diminution, amounting even to total abolition of faradic excitability. This also holds good, mutatis mutandis, for those muscles of the body that are accessible, which as a rule are supposed to be excited at the points of entrance of their motor nerves.

The behavior of the nerves and muscles in response to opening and closing, and to the continuous passage of the galvanic current, is termed *galvanic excitability.* The actions of the two poles upon the living body are best investigated separately, both on grounds of physiological as well as of physical convenience, and cannot here be more fully discussed (polar method). The nerves and muscles to be investigated are brought into connection with one—the exciting pole, whilst the other—the indifferent—pole is applied to some indifferent part of the body (preferably to the sternum). Inasmuch as the exciting pole is sometimes the anode and sometimes the cathode, and the current is sometimes closed and sometimes opened, a series of reactions are obtained (namely, upon cathode closure=KaS, and cathode opening=KaO, upon anode closing=AnS, and anode opening=AnO), which together constitute *the law of contraction of motor nerves and muscles.*

The law of contraction of motor nerves rests upon the two following propositions:—The cathode produces *chiefly* (or, strictly speaking, exclusively) contraction on closure of the current, the anode *chiefly* (or exclusively) contraction on opening the current; the stimulus of the cathode is stronger than that of the anode. From this may easily be deduced the following formulæ of reaction with different strengths of current:

Lowest grade, a weak current being employed: KaSZ;[2]—the cathode produces simple contraction upon closure of the current; no reaction from the anode.

Intermediate grade, the current being of medium strength: KaSZ′, AnSz,[3] and AnOz;—the cathode causes stronger contraction upon closure of the current, but no contraction when it is opened; the anode, on the other hand, causes feeble contraction both when the current is closed and when it is opened—the degree of contraction being nearly the same in both cases, though sometimes the one and sometimes the other may be somewhat the stronger, or may show itself more promptly.

Highest grade, with a strong current: KaSTe, AnSZ, AnOZ and KaOz;—upon closure of the current the cathode produces a tetanic tonic contraction, while on opening the current feeble contraction occurs; with the anode lively contraction occurs both on closing and opening the current.

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[1] See in regard to details: *Erb*, zur Lehre von der Tetanie nebst Bemerkung über electrische Erregbarkeitsprüfungen u. s. w. Archiv für Psychiatrie und Nervenkrankheiten. Band IV., 1873.

[2] Z stands for the German word *Zuckung*=contraction; Te for tetanic contractions; S. for *Schliessung*=closure of the current; O. for *Oeffnung*=opening of the current; Ka and An, for cathode and anode respectively.

[3] The accentuation (Z′) simply refers to the stronger degree, the small z to a lower degree, of contraction.

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It thus becomes apparent how the various contractions must succeed one another, as the strength of the current is increased, and how requisite it is that the relative intensity should be the same, if we are desirous of estimating qualitatively and guantitatively the normal excitability of the motor nerves for the galvanic stimulus. With a little practice, and if proper precautions be taken, this law of contraction will be found to be essentially the same for all accessible motor nerves.

The law of contraction for the muscles is identical with the one just given; it depends, however, upon the excitation of the intra-muscular nerve endings, though it is probable that the muscles themselves possess a proper excitability which only becomes apparent when the nerve terminations are excluded, as occurs in pathological conditions.

Deviations from this normal law of contraction, on the application of the galvanic current, frequently occur in pathological conditions, the changes being both qualitative (variations in the form and mode of contraction) and quantitative (increase and diminution of galvanic excitability). We shall refer to this in the several chapters.

1. On Spasm or Convulsion in General.

Romberg, Nervenkrankheiten, I. p. 335. 3. Aufl. — *Hasse*, l. c., p. 126. — *A. Eulenburg*, funct. Nervenkr., p. 623. — *J. Chr. Clarus*, Krampf in path. u. therap. Hinsicht. Leipzig, 1822. — *L. Fleckles*, die Krämpfe an allen ihren Formen. Wien, 1834. — *Natanson*, Beitr. z. Physiol. Diagnostik der Krämpfe; mitgetheilt von Bergson. Deutsch. Klin. 1860, No. 25. — *Nothnagel*, Entstehung allgem. Convulsionen vom Pons u. v. d. Medull. oblong. aus. Virch. Arch. Band 44. — Z. Lehre vom klon. Krampf. ibid. Bd. 49. 1869. — *Hitzig*, über Reflexerregende Druckpunkte. Berl. klin. Woch. 1866, No. 7. — Untersuch. z. Physiologie des Gehirns. Reichert u. Dubois' Archiv. 1870, 1871 und 1873.— Ueber die Auffassung einiger Anomalien der Muskelinnervation. Arch. f. Psych. u. Nervenkr. III. pp. 312 u. 601. 1872. — Ueber einen interess. Abcess. der Hirnrinde. ibid. III. 1872. — *Wernher*, Verletzung der linken Grosshirnhälfte. Virch. Arch. Bd. 55. 1872. — *Benedict*, Nervenpathol. und Electrotherapie 1874. — *Ranke*, über die krampfstill. Wirkung des const. el. Stroms. Ztschr. f. Biolog. II. 1866. — In addition to these, consult the works of v. *Graefe*, and of *Remak*, and the text-books on Electrotherapeutics.

Any attempt to give an exhaustive account of all the relations that may occur in the vast category of *spasms* or *convulsions* is here quite impracticable, and does not lie within the scope of this work; it belongs rather to the domain of general pathology. The conditions here are much more complicated, and differ from each other more widely than in the neuralgiæ, for example, the pathology and the therapeutics of which may easily be described in a general manner, and which, besides, possess a very strong practical interest. Convulsions cannot be regarded from any single point of view, and do not represent any single form of disease. The subject of convulsive diseases is, it is well known, very obscure, and we possess little positive knowledge—scarcely, indeed, more than mere assumptions—in regard to their most important and essential relations.

A few observations only will here be made, which, however, may prove useful in facilitating the comprehension of the several forms, and the account now to be given of them.

The very definition of the term "spasm" presents considerable difficulties on account of the numerous and manifold phenomena which are included under this term in ordinary language. The best interpretation of the word includes *all abnormal muscular contractions*, which are either occasioned by pure pathological stimuli, or in which the amount of contraction is obviously disproportionate to the amount of the physiological stimulus. Convulsive muscular contractions are thus abnormal, either in consequence of being caused by new, unusual, pathological excitations of motor ganglion cells, nerve fibres or muscles, or in consequence of the fact that an abnormal increase has taken place in the motor excitations supervening upon physiological stimuli, like those of the will, of reflex or automatic excitations. There is thus almost always in spasm an increase of motor activity, in other words, an increase of its visible expression—muscular contraction.

The modes in which these pathological contractions of muscle make their appearance are extremely various, and very different forms of spasm may therefore be distinguished. Many forms of spasm occur in practice which often present but little similarity to one another, and the attempts that have been made to give a general description applicable to all these various forms have not been hitherto very successful.

The most general division that can be made is into *tonic* and *clonic* spasms. By the term *tonic spasms* are understood persistent and almost equable muscular contractions of great intensity, lasting for a considerable period (minutes, hours, days), by which the limbs are kept in a state of rigidity. By the term *clonic spasms* is understood the rapid succession of contraction and relaxation of the muscles, or the rapid alternation in the contraction of various groups of muscles, by means of which different parts of the body are often kept in a state of continual and frequently very lively movement. The essential difference between these two forms of spasms is perhaps this, that in tonic spasms the several motor excitations are so closely approximated to one another in point of time, that they coalesce to produce a continuous tetanic contraction; whilst in clonic spasm the several excitations are separated from one another by intervals of sufficient duration (varying also in intensity and in the part affected) for each excitation to be separately recognized.

This division is not, however, exhaustive, because it does not include all the forms; a group of convulsive movements must still be formed, which includes the co-ordinated but involuntary movements (static convulsions, compulsory movements, *co-ordinated spasms;* which last appears to me to be the best term for this group of spasms, since the definition of static spasms or convulsions varies considerably with different authors).

Among the tonic and clonic spasms, there are several subordinate yet well-defined forms, the more important of which may here be briefly mentioned.

The mildest form of clonic spasm is quivering or tremor, which consists of slight contractions of particular muscles and groups of muscles, following one another at short intervals, or of weak alternate contractions of antagonistic muscles, whereby slight trembling or oscillatory movements of the limbs are produced. The higher degrees consist of more powerful though brief contractions of the muscles, which cannot be checked, and which occasion very manifest trembling and even distinct quivering of the limbs (paralysis agitans).

Still more extensive are those convulsive movements termed *convulsions*, which consist of energetic contractions in particular muscles or groups of muscles, occasioning lively movements, which succeed one another at short intervals (twitchings of the face, movements of the head or body, startings of the limbs). If these convulsions affect the majority of the muscles and occur in rapid succession in different parts, leading to brisk, extensive, and irregular movements of the trunk and limbs, they are spoken of as general convulsions; they constitute the principal symptom of epileptic, uræmic, and hysterical convulsions.

The simplest form of tonic spasm is the so-called *cramp*—a persistent, violent, and painful contraction of some single muscle or particular group of muscles, examples of which are seen in cramp of the calf of the leg, trismus, and tetanus. If this form of spasm affect a majority of the muscles of the body, and recur in paroxysms of longer or shorter duration, *tetanic cramps* are said to be present (as in tetanus).

A peculiar form of cramp may be observed in the uniform but not very strongly marked stiffness of many or all the muscles of the body that occurs in catalepsy; the muscles here remain immovable, in a condition of moderate contraction, the will having scarcely any influence upon them, though the resistance they offer can be overcome with tolerable facility by passive movements (waxen pliability).

The term *contracture* is employed to indicate the persistent and inextensible shortening which lasts for weeks or years, and is frequently associated with certain trophic disturbances of the muscles; this condition may be limited to a few muscles, but usually affects entire groups of muscles, as the flexors or extensors of particular joints.

Movements of a higher order may also be excited in a cramp-like manner by excitation of the motor centres, leading to complicated movements or to purposive combined movements; these, associated and co-ordinated in a cramp-like manner in opposition to the will of the patient, often occur automatically. Such movements are termed *compulsory movements*, *co-ordinate cramps*, and examples of them are seen in cases where there is a disposition to walk to one side or in a circle, to perform certain movements with the arm or head, etc.

Lastly, *cramp-like associated movements* occur, which accompany movements performed voluntarily, and disturb them or make them uncertain and purposeless, or which even occur upon the simple desire to make a movement; these are especially seen in chorea minor.

Many other forms of cramp are occasionally seen in practice which are not easily included in the several forms mentioned above; those that have been alluded to, however, are much the commonest and most important.

Spasms must of course in all instances be induced by irritation of the motor apparatus, and such irritation may result either from *increase of the strength of the stimulus*, so that a motor effect is produced, corresponding to the abnormal strength of the stimulus, *or from increase of the excitability of the motor apparatus*, so that a motor effect is produced far exceeding the amount of the stimulus applied. All spasms may be referred to one or other of these causes; augmentation of the excitability, however, probably plays a more important part in the pathology of spasms than increase of the strength of the stimulus. The question next presents itself at what part of the motor paths the stimulus must be applied, in order to cause cramp, and after this the further question, what is the nature of this stimulus, whether momentarily, or continuously, or intermittingly applied, or whether it be mechanical or chemical in its nature.

The most cursory as well as daily observation teaches that the stimulus may either be applied *directly* to the motor apparatus and nerves at some point of their course, or *indirectly* and in a *reflex manner;* and it appears that a natural division of convulsive affections into two groups may thus be made, which present certain differences in their mode of origin, in their extent, and in their anatomical localization. When Natanson therefore maintains that direct cramps always appear in the form of persistent, tonic, violent and painful muscular contractions, affecting certain anatomical groups of muscles supplied by motor nerves (particular nerves or definite plexuses), whilst indirect spasms appear in the form of clonic short alternating and painless muscular contractions, which affect physiologically associated muscular groups, such as the flexors and extensors of the extremities, the muscles of respiration or the like, he makes a very well-founded statement, and one which materially elucidates the theoretical considerations that have been advanced upon the subject. It must not be forgotten, however, that the facts hitherto ascertained by no means support so simple an explanation of the phenomena; numerous further observations are undoubtedly required to answer this extremely important and, in practice, very significant point.

Spasms which originate from direct excitation can of course be produced by an irritation applied to the motor nerves at any point between the periphery and the centre; reflex spasms, on the other hand, can only result from some cause acting on the central organ, through the intervention of the gray substance.

Direct spasms may originate *by irritation of the muscular fibres themselves*, or *of the motor terminal plates in their interior.* The so-called fibrillar contractions, as well as certain forms of muscular contracture and rigidity which occur in consequence of paralysis, with consecutive anatomical changes in the muscles, belong to this category.

They may originate by *irritation of the peripheral motor channels of conduction*, up to their entrance into the spinal cord or brain. Traumatisms, such as may be accompanied by compression and tearing of the nerves, gunshot wounds, and inflammation of the motor nerves, constitute the commonest causes of this kind. The spasm is then strictly limited to the affected motor path, is for the most part of a tonic character, and is ultimately associated with corresponding sensory and vaso-motor disturbances. They may be produced, in the third place, by *irritation of the channels of conduction and of the motor* (*reflex*) *centres in the spinal cord.*

To this category belong the contractures, conditions of muscular tension, spasms, etc., that are seen in myelitis and other spinal diseases, the cramps observed in tetanus and similar spinal neuroses. The precise mode in which these forms of spinal convulsions are produced is still obscure; perhaps in many cases some direct irritation of the motor channels of conduction occurs in the antero-lateral columns and in the gray substance, or there may be some irritation and increase of excitability of the motor central apparatus lying in the gray substance, or of those ganglion cells which are capable of transmitting motor impulses through reflex action.

We here generally have to deal with spasms of well-defined extent (affecting for instance both lower extremities, the flexors or extensors, the paths for which lie in close proximity in the spinal cord). In these cases concomitant spinal phenomena are almost always present.

Again, they may be produced by *irritation of the conducting paths and central apparatus in the brain.* The relations here are extremely manifold and complicated. In the first place, the simple motor channels of conduction in the crus cerebri (Gehirnstamm) and in the corona radiata, may be subjected to pathological irritation, and it is well known that in certain diseases of these structures, spasms do not unfrequently occur. In the next place, the motor centres, which Fritsch and Hitzig have demonstrated in the gray cortex of the anterior lobes, enter into consideration, for the recently published interesting observations of Hitzig and Wernher show that in men also the excitation of these centres through pathological conditions may lead to the occurrence of limited peripheral spasms. A very similar and now intelligible observation is recorded in the work of Debrou, upon spasms of the facial muscles ;[1] here belongs also a recently published observation by Goldstein (Schmidt's Jahrbücher, Band 161, Heft 2). In this connection should be mentioned pathological excitations of certain automatic centres (centres, for instance, of inspiration and expiration, those for the acts of laughing, yawning, etc.), which may undoubtedly be the points of origin of certain forms of cramp. Still more complicated are the relations that exist between the manifold connections of the motor paths, which are undoubtedly situated in the central organs, combinations which render possible the coincident and quite involuntary innervation of numerous muscles and groups of muscles which occurs in every voluntary movement, however simple it may be. Convulsive movements may result also from pathological changes in these paths of conduction. It is probable that the pathologically associated movements, which arise from collateral extension of the voluntary motor excitation to paths of conduction not usually employed, even though anatomically preformed, are referable to these communications, and Hitzig has sought to attribute hemiplegic contractures to pathological changes in them. Still more difficult and obscure are the relations in certain forms of spasm, the cause of which we have likewise to seek in the brain, and which are termed co-ordinated spasms, compulsory movements, and the like. It is scarcely requisite, however, to enter here into the consideration of these extremely obscure conditions, which may, besides, vary so greatly in significance, and which will be discussed in other parts of this work.

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[1] Archives Générales, 1864, case 5 ; right-sided spasm of the facial and masticatory muscles with aphasia ; small apoplectic clot in the left frontal lobe.

Lastly, Nothnagel has demonstrated the existence of a special *convulsive centre*, that is to say, a circumscribed spot in the central nervous system, by the direct or indirect irritation of which convulsions can be produced, as in epilepsy. This spot lies in the pons, and is probably situated at the point where the motor nerves of the antero-lateral columns first terminate in ganglion cells. Reflex irritation of this convulsive centre may be best and most certainly accomplished by irritation of a circumscribed spot on the floor of the fourth ventricle (on both sides, at a little distance from the middle line), the precise boundaries of which, however, have not yet been exactly determined in the case of man.

Convulsions, arising in a reflex manner, take place through the intermediation of the gray substance of the central organs, even though the focus of excitation may be at some distance. The simplest forms of reflex convulsions are produced through the spinal cord, the more complicated through the medulla oblongata and the brain, where the sensory and motor fibres enter at various points into communication with one another by means of the gray substance. Reflex spasms certainly form a very important and large group amongst convulsive affections, and innumerable cases differing considerably *inter se* belong to this category. Like reflex processes generally, they may arise in various modes, namely:

By increased irritation and augmented excitability of the *centripetal (sensory) fibres.* This is the most common origin of reflex convulsions, and every conceivable irritation of any part of the body may, in this way, give rise to such convulsions; thus, for example, they may arise from irritation of the sensory nerves in consequence of wounds, inflammations and other irritations of the nerves, neuralgiæ and hyperæsthesiæ; from irritation of the skin, and especially of the mucous membranes, by excitations of all kinds consequent on inflammations, ulcerations, foreign bodies, concretions, and worms; and to these may be added, the forms of cramp included under the terms blepharospasm, cystospasm, vaginismus, and those resulting from diseases of the generative organs, intestines, etc. All these forms of convulsion follow the general physiological laws of reflex processes; they are limited, in the first instance, to the muscles that are nearest to them, then spread to the more remote, and may ultimately involve numerous groups of muscles.

By increase of the excitability of the reflex centres in the spinal cord and brain. This may be caused by various forms of disease as well as by the toxic influence of certain poisons, such as strychnine; in such cases, as is seen in tetanus and myelitis, ordinary physiological sensory excitations are sufficient to produce convulsions. A lower degree of this increase of excitability probably plays an important part in the production of many reflex spasms. This constitutes what is termed a predisposition to spasms, or convulsibility, and leads to an outbreak of a convulsive nature when any lively sensory influence is experienced, as is the case in hysteria.

By removal of the activity of those cerebral centres which inhibit reflex actions. If the activity of these centres be removed (by destroying them, or by section of the conducting fibres which pass to the spinal cord), the reflex actions, performed through the intermediation of the spinal cord, are increased in activity, and may themselves acquire a convulsive character. But little further is known in regard to these processes in human pathology.

That a diminution of the resistances to conduction, in the centrifugal or motor nerves, is capable of producing an exaltation of the reflex acts, may be assumed on *à priori* grounds. It nevertheless appears questionable whether, by this means, with physiologically normal sensory stimuli, and with normal excitability of the reflex centres, any spasmodic movements that may occur, can be included in the category of reflex convulsions. Our present knowledge has hitherto done little to explain the mode of origin of such convulsions.

In reference to the kind of excitation, and to the question in what mode pathological excitations attack the different points of the motor apparatus, and in regard to the nature of the stimuli that excite cramp, we are still completely in the dark. It is obvious that the changes in the motor apparatus are of a very delicate nature; where coarse anatomical changes are present, paralysis, and not cramp, is the invariable consequence. At the same time, as daily experience shows, convulsion and paralysis may occur in the same nerve territories, but this is readily explicable on the same grounds as those which hold good for anæsthesia dolorosa, and in such cases cramp is produced at those points of the motor path which have not undergone any considerable change in their structure.

The mode in which the stimuli act which excite the various forms of convulsion, and which lead to tremor in one case, to spasm in another, and to tetanus and clonic convulsion in a third, has led to the annunciation of very different hypotheses, and, after all, very little is known in regard to it. Tonic convulsion chiefly occurs where direct stimuli act on some part of the motor paths (Natanson); clonic convulsion, on the other hand, appears to be caused by indirect excitation. This, however, can scarcely be admitted unrestrictedly for all cases; at the same time it is in accordance with the fact that Nothnagel has found the presence of gray matter to be indispensable for the production of clonic convulsions; the transmission of a stimulus through the gray matter appears to act in such a manner that continuous irritation only gives rise to intermitting muscular movements. This indicates that in the greater number of clonic convulsions the gray matter of the central organs probably participates.

Here also, on this difficult point, our information is very imperfect and hypothetical; the little that we do possess relates to some of the *more immediate causes of convulsions*, the knowledge of which affords us not a few glimpses into the pathogenesis of certain forms of convulsion, although we are, it is true, comparatively ignorant in regard to the more delicate details of their mode of action.

The simplest of these causes are *mechanical stimuli*, which, as experience shows, may induce convulsions in the most various parts of the motor apparatus; section, contusion, laceration of peripheral nerves, are all accompanied by convulsion. Convulsions of greater or less extent, according to the plane of the part injured, are also called forth by section of the spinal cord; and these convulsions are tonic when they are produced by direct irritation of the motor paths; clonic, when they are produced in a reflex manner through the intermediation of the gray substance. Mechanical injuries of various parts of the brain may also give rise to cramps. Nothnagel made his researches upon the convulsive centre by means of mechanical stimuli. The formation of apoplectic clots is usually accompanied by convulsive movements.

Chemical stimuli are often not less active; a great variety of substances, especially certain poisons, can so modify the motor apparatus as to cause convulsions, either directly or by reflex action. For example, strychnine, ergotine, and numerous other alkaloids, alcohol, lead, and mercury.

There are, in addition, certain *disturbances of the circulation* which we must consider as not unfrequent causes of spasm and convulsion. It is well known that extreme anæmia, at least of certain segments of the brain, and especially, as it would appear, of the convulsive centre, can give rise to widely extended and violent convulsions. (This, however, does not occur with anæmia of the spinal cord, as is shown by Stenson's experiments.) The same holds true of extreme *venous hyperæmia and stasis*, which, when affecting the brain, are also capable of giving rise to convulsions (Landois, Hermann). In both cases the irritation is probably due to an accumulation of carbonic acid, or other products of disintegration, in the tissue of the brain. By means of these facts we are better able to understand how convulsions originate in vaso-motor neuroses. A sudden anæmia, caused by contraction of the vessels, is undoubtedly the exciting cause in many forms of convulsion, and it is possible that many sensory excitations act in this indirect mode by producing vaso-motor spasm.

Alterations of the blood may lead, in various modes, to the production of convulsions, partly in consequence of the blood containing, as in uræmia and cholæmia, toxic substances, partly in consequence of its defective qualitative composition, leading secondarily to trophic disturbances in the nervous system, and thus to convulsion, or at least, to a strong predisposition to convulsion, as in hydræmia, chlorosis, general cachexia, etc.

The following *anatomical changes* of the motor apparatus are not unfrequently accompanied by convulsions : inflammation, neoplastic formations, softening, extravasations of blood with their consequences, etc.; at the same time we know nothing of the precise mode in which these effects are produced.

We are not unfrequently compelled to admit the existence of *trophic disturbances of an impalpable nature*, in order to obtain a plausible explanation of the origin of many forms of convulsion, as of those that occur in epilepsy, chorea, and hysteria. It is upon such delicate disturbances of nutrition that that condition depends which is usually termed convulsibility, or a *predisposition to convulsions.*

This predisposition plays also a prominent part in the production of very numerous forms of convulsion ; it may be congenital, inherited, or acquired through the action of various conditions and diseases. It may coincide in many cases with a general neuropathic predisposition which exists in many families, and is a fruitful source of many neuroses. Youth, the female sex, many diseases incident to the period of development, and especially chlorosis, appear strongly to favor its activity. We must, undoubtedly, seek for the essence of this predisposition in the finer trophic disturbances of the motor apparatus, though in regard to the peculiarities of these, we know but very little. Some facts that are now known render it probable that such trophic disturbances can in many cases be recognized by alterations (especially by increase) of the electrical excitability.

Lastly, those disturbances which are least palpable, but which not unfrequently occasion well-marked and severe convulsions, are those of a *psychical* nature, particularly the powerfully acting and depressing emotions, as, for example, anxiety, fear, anger, horror, witnessing others in convulsions, etc., which all play a very important part in the etiology of convulsion. In what method, however, these causes operate, whether they produce a direct alteration in the motor apparatus, or whether they act indirectly by some influence exerted upon the vasomotor nerves, or in a reflex manner, we are perfectly ignorant.

In treating of the several forms of convulsions we shall still have to mention many special causes.

General Symptomatology.

The distribution of convulsions in the body may be extremely various, the diversities being dependent on the nature and particular part of the nervous system affected by the cause. With local peripheric excitation the spasms may be limited to single muscles or to particular groups of muscles ; it must not be forgotten, however, that very isolated and circumscribed forms of convulsion may have a central origin, which is easily explicable when the course of the fibres in the brain is taken into consideration. If the spinal cord be affected as a whole, the greater part of the muscles of the body may be attacked, including even those supplied by the motor portion of the trigeminus. Lastly, if the convulsive centre be affected, the most general distribution of the convulsions may be anticipated. Between the two extremes all possible degrees exist, in accordance with the anatomical seat of the disease. Thus we may have limitation to a single muscle, as, for example, a particular muscle of the eye, or the sterno-cleido-mastoid ; or to a single nerve, as to the facial or accessory ; or to a plexus or to one extremity, as occurs in paralysis agitans and paralytic contracture ; or to one-half of the body, as occurs in unilateral convulsions consequent on disease in one hemisphere of the brain ; or to the lower half of the body, as in spinal diseases ; or, lastly, almost all the muscles of the body may be affected, as is seen in tetanus and epilepsy. Not unfrequently certain groups of muscles only are affected, which ordinarily act in an associated or synergetic manner, or which are under the control of a special centre, examples of which are seen in compulsory movements, in spasms of the respiratory muscles, and in spasms of the muscles by which yawning is effected.

From all this it is obvious that definite conclusions regarding the seat of the lesion may often be drawn from a consideration of the parts that are affected.

But few observations of a general nature can be made in regard to the mode of onset of spasmodic affections. It varies extremely in different instances, and will be given more exactly when the different forms are severally described. Convulsions very frequently occur in distinct, well-defined, and characteristic paroxysms, which often pursue a typical course, and recur more or less regularly, with intervals of longer or shorter duration. They often appear also, however, quite irregularly, being induced by certain external causes, or they may be associated with voluntary movements, or may occur quite spontaneously, and may then last for a very variable period, or even become permanent. In addition to pathological muscular actions, a series of other phenomena are not unfrequently observed, which complete the picture of the disease, and which we may divide into concomitant symptoms and consecutive symptoms, or sequelæ of the affection. Amongst the *concomitant phenomena* are comprehended those which originate from an extension of the disturbance to other parts of the same nerve, or to other nerves, whilst under the term *consecutive symptoms* are included those which represent the effects of the convulsion itself, that is to say, of the spasmodic shortening of the muscle.

The concomitant phenomena are by no means so constant, nor are they present in so characteristic a manner, in spasmodic affections as in neuralgiæ. They present for the most part the character of accidental symptoms occasioned by the same causes of disease as those by which the spasms have been produced.

In many cases the concomitant symptoms are absent, as in strabismus and in simple convulsive tic; the spasm is here the only direct subjective and objective symptom of the neurosis, and, in addition, the consecutive symptoms can alone be observed.

In most cases, however, various concomitant symptoms are observed, namely:

Motor disturbances, the most common of which is seen in the diminution of the power of the will over those muscles that are spasmodically affected,—a diminution which may amount even to complete paralysis. This may come to pass in the most various ways, for the particulars of which the several forms must be referred to. For the most part there is an inhibition of conduction caused by the primary disease in some part of the motor path situated at a higher plane. A kind of irradiation of the motor stimulus also takes place; at the height of the convulsion associated movements are seen to occur in more or less numerous groups of muscles, obviously in consequence of the extension of the irritation upon the various inter-communicating paths.

Sensory disturbances. Pain is very frequently observed as an accompaniment of spasms, not only in *the contracted muscles themselves*, but also proceeding or radiating from them; this is especially noticeable in simple cramps, in tetanus, etc., though on the other hand it is often absent in many forms of spasm. Its mode of origin is still a subject of dispute; it cannot, however, be doubted that the pain is due to an irritation of sensory muscle-nerves, caused by muscular contraction,—an irritation of those very nerves which are the conductors of both muscular and electro-muscular sensibility. That the pain is not caused by mechanical compression of sensory cutaneous nerves running between the muscles, is shown by the absence of excentric sensations of pain in the skin, as well as by the fact, of which every one is aware who has ever suffered from cramp in the calf of the leg, that the pain is unquestionably limited to the firmly contracted muscle. A very decided sensation of *fatigue* and *exhaustion* is often observed in the affected muscles after the cessation of the cramp. Not unfrequently also that well-known peculiar *muscular pain* is experienced which often occurs in the muscles of healthy people for about twenty-four hours after violent and unusual muscular efforts, and which is felt both on active contraction and when pressure is applied to the affected muscles.

Pain also occurs in various nerves when sensory fibres are excited coincidently with motor fibres, as in affections of mixed nerves or of the central organs, or when reflex convulsions occur in neuralgia. This depends upon the nature and kind of the primary disease. The same is true in regard to the frequently coincident symptoms of *formication, numbness, or well-marked anæsthesia.* The occurrence of these symptoms depends entirely upon the circumstances of the particular attack, and has nothing to do with the spasm *per se.*

Vaso-motor and Secretory Disturbances. These are frequently wanting, and in many forms of spasm are altogether absent. When they are present, their relations to spasm appear to be very slight, and they may be regarded rather as symptoms of the same disease which produces the spasm. Vaso-motor spasm or paralysis, indicated by pallor or redness of various parts of the skin, has often been observed to be a precursor of certain convulsive paroxysms (*e. g.*, epilepsy); and so also abnormal sensations of cold or heat, with the corresponding disturbances of the circulation, are frequently associated with convulsive attacks. If the disease affect coincidently vaso-motor nerves, whether these are situated in the peripheral nerves or in the central organs, corresponding localized vaso-motor disturbances, such as paleness, redness, cyanosis, may occur.

The *perspiration* is the secretion which is most frequently altered. Profuse perspirations occur both in epilepsy and in tetanus, and sweating on one side of the body has been frequently observed in unilateral epilepsy. Under the term of *urina spastica*, that clear and abundant secretion of urine is indicated which has not unfrequently been observed after severe attacks of convulsions. No instance of augmented salivary secretion has been observed in cramp of the facial muscles.

The *trophic disturbances* are, in the majority of cases, very insignificant. It is often quite surprising what slight alterations occur in the volume and nutrition of muscles affected with the most severe and obstinate forms of convulsion. In many cases neither hypertrophy nor atrophy is visible. These effects, however, do undoubtedly occasionally occur. Hypertrophy of the muscles which have been affected with convulsion, is very rare (Bell). Atrophy is more frequent, and may become tolerably well marked. Serious motor disturbances (paralysis) are then usually present, and the atrophy appears to be the consequence of this, rather than of the convulsion. Considerable anatomical changes are then commonly observable in the muscle, namely, hypertrophy of the connective tissue with atrophy of the muscular fibres, and not unfrequently a deposit of fat—changes which are constantly present in contractures that have lasted for many years.

Well-marked trophic disturbances, occurring in the skin, nails, and other tissues, are only observed when there is serious lesion of the trophic and vaso-motor nerves, and are then quite accidental symptoms. In such cases a high degree of anæsthesia and paralysis is almost always present.

Psychical disturbances are not unfrequently associated with spasms, though they are not usually directly dependent upon them. The connection between the two symptoms is still in many cases extremely obscure. In some of the cases the psychical disturbances are simple co-effects of the same cause; in others, the serious damage to the nervous system, occasioned by violent attacks of convulsion, or the disturbances of the circulation caused by such attacks, may gradually lead to the occurrence of psychical symptoms; or, in many patients, a hypochondriacal condition may be gradually induced in consequence of the existence of the convulsive affection. These relations, however, will have to be investigated more closely in each individual case.

Disturbances of the general health are, upon the whole, only rarely observed; and when they are present, they are, for the most part, consequences of the primary disease, and not related in any way to the convulsion. More minute details will be given in speaking of the several forms of convulsion. It may, however, be stated here that the spasms, even when affecting many groups of muscles, are not capable of producing any considerable elevation of the temperature of the body, and that, consequently, the pyrexia that accompanies so many forms of convulsive disease, must have another origin. Sufficient evidence exists to demonstrate this fact satisfactorily.

The sequelæ of convulsions are, for the most part, purely mechanical, and result exclusively from the abnormal muscular contraction. It is obvious that in accordance with the seat of the convulsion they may be extremely various. Thus there occur disturbances in the functions of the organs of special sense (strabismus, blepharo-spasm), impairment of the mobility of the joints, disturbances in the co-ordinated movements of the body, abnormal position of the head and limbs, curvature of the spinal column, alterations of the articular extremities of the bones, displacements and subluxations of the joints; and, in addition, difficulty of breathing, difficulty in passing water, in defecation, and in taking food. General trophic disturbances, psychical disorders, and the like, may thus arise in a secondary manner. All details on these points must be reserved till the several forms of convulsive diseases are under discussion.

In regard to the behavior of *diseased motor nerves and muscles under electrical stimulus*, nothing positive can be said. The marked and diagnostically important changes that we shall describe further on as characteristic features in the different paralyses, do not here occur. The methods of research hitherto employed for the recognition of the more delicate, and especially of the quantitative changes of electrical excitability, are so unsatisfactory in their results, that most of the statements on this point can only be accepted with reserve. In many cases scarcely any changes of electrical excitability are to be found; in some, however, there is diminution; in others, exaltation. In a few cases of tetany I have found great exaltation of excitability, especially for the galvanic stimulus; but these cases I shall describe at greater length hereafter under the head of Tetany. These few facts do not at present admit of any generalization. Moreover, the electrical method of research has hitherto proved of but little value in the pathology and diagnosis of convulsions in general.

Lastly, a series of phenomena require still to be mentioned, which attract attention in many forms of convulsion, and which appear to be of great importance in the pathology as well as in the treatment of these convulsions: I refer to the existence of *pressure points*. Experience has shown that in a series of convulsive attacks pressure upon certain points puts a stop to the convulsions when present (von Graefe, Remak), whilst in other cases the convulsive attacks are brought on by pressure made at particular points (Hitzig). The pressure points inhibiting convulsion have been especially observed to exist in cases of facial spasm, and it has been ascertained that these, for the most part, correspond, like the painful points in neuralgiæ, to the various branches of the trigeminus. These spots are also not unfrequently sensitive to pressure. It has been observed that the same effects are produced by pressure on very remote points, as the vertebral column and the joints of the hand. Von Graefe drew a distinction between primary and induced pressure points. The former, as he considered, constitute the chief point of origin of the disease, and appear to stand in the most direct reflex relation to the primarily and most strongly affected muscles; pressure over these completely arrests the entire convulsion. The induced pressure points, on the other hand, make their first appearance when the disease has become established, and are discoverable in more or less remote nerves; pressure made upon them requires more time to act, and never completely arrests the convulsion, but rather leaves the proper point of origin of the convulsion unaffected. Similar points have also been observed in other forms of convulsion than those affecting the face. These phenomena can only be explained on the supposition that they are the result of reflex processes. It is possible that pressure upon particular nerve trunks may prevent the transmission of sensory stimuli proceeding from the areas of their distribution, and thus arrest the excitation of reflex convulsions; hence the spasm is temporarily inhibited. But it is more probable that reflex inhibitory influences are here brought into action, which obviously proceed from energetic sensory impressions, and which have recently been carefully investigated by physiologists. It thus becomes intelligible how an inhibition of the convulsion can be produced by pressure on various points. Which of these theories is correct, and whether each cannot be separately justified, must be decided by further facts bearing upon the point.

An interesting case of pressure points producing reflex actions has been published by Hitzig. In this case no other explanation could be given than that the actions were reflex. Both kinds of pressure points are of considerable importance in regard to treatment, because not unfrequently a favorable modification of the convulsions may be effected through them.

In regard to the general *prognosis* of spasms, but little is to be said in view of the great variety in the mode of origin of the numerous forms of spasm. The general impression obtained is, that spasms are to be regarded as severe and obstinate affections. It is only in the simplest cases of reflex spasms, or of convulsions dependent upon direct irritation that a favorable prognosis can be given. In the majority of other forms, especially if the spasms have become an habitual thing, and are accompanied by a well-marked neuropathic predisposition, and in most idiopathic forms, the most practised physician can only give a very guarded prognosis, because even apparently slight cases often unexpectedly bid defiance to all methods of treatment. At all events, experience teaches that the greater number of the forms of spasm are cured with difficulty, and that relapses are very common.

All details must be reserved for the description of the special forms. It may, however, here be mentioned, that the prognosis is rendered unfavorable when the patient presents a well-marked neuropathic predisposition, when the disease is of central origin, when organic lesions of the brain or spinal cord exist, and when it is impossible to remove the cause; advanced age of the patient and long duration of the disease, are also unfavorable circumstances.

On the other hand, the prognosis is favorable when the causes are of a simple nature and easily removed, when the lesion has a peripheral seat and the attack is of recent date, and when the patient is young and healthy.

General Treatment.—Spasmodic diseases are amongst the most unsatisfactory objects of medical treatment, and subject both the patience of the sufferer and the ingenuity and perseverance of the physician to a severe trial. There is little to be said in regard to the rational treatment; but this is not to be wondered at, when our ignorance of the finer processes taking place in convulsive diseases is considered. Treatment is purely empirical, and is often also unsuccessful. It frequently happens that well-approved measures fail to act, whilst a plan of treatment made in the last extremity, and without any expectation of good results, proves successful.

Nevertheless, in every case the treatment should be conducted as far as possible on rational principles, and above all it should be directed to the primary cause. Unfortunately, in many cases no cause can be discovered, whilst in others the spasm persists after the removal of the cause, the cramp having become habitual. Causal treatment is most efficacious in reflex spasms; and in these cases a careful investigation of the whole sensory nervous apparatus, and of all those organs from which reflex excitation may possibly proceed, is of the utmost importance. Organic lesions must be cured; conditions of irritation must be quieted; and foreign bodies, parasites, and the like must be removed. If pressure points be discovered, treatment must be attempted through them, as by mechanical and electrical treatment, and well-devised sections of nerves often give excellent results under these circumstances.

In convulsions caused by organic diseases of the central or peripheric nervous system, these diseases must be carefully treated on the principles of special pathology.

The *treatment of the general predisposition*, of the convulsibility, requires special attention, and here all measures acting as tonics to the nervous system must be taken advantage of. These are in many cases alone sufficient to effect a cure, and are often more appropriate in convulsions than in other neuroses; at all events, they can effectively aid the action of other curative means. The most important facts in regard to the tonic method of treating the nerves have already been mentioned in the section on treatment of neuralgiæ in general.

Dietetic measures also form an important part of the treatment, to which, in almost all forms of convulsion, due attention should be paid. Besides tonic treatment of the nerves, well-regulated measures should be adopted in regard to the proper kind of food and drink to take, what baths to use, how much exercise and how much rest to take. Moderation in eating and drinking, and in the use of luxuries, avoidance of all exciting and debilitating pleasures, of social gatherings protracted to late hours of the night, of gaming, and of sexual excitations and excesses, avoidance of all mental disturbance, injurious excitation, excessive stimulation of any of the sensory organs, as, for example, by music, must be prescribed. Efforts must be made also to strengthen the will by education, precept, and example, which may be materially promoted by the rational use of various kinds of gymnastic exercises, in accordance with the idiosyncrasy of the patient and the peculiarities of his case. To one patient we may recommend chamber gymnastics, to another the Swedish movement cure, and to a third German gymnastics or Alpine climbing. These general observations will of course only serve as hints in determining the appropriate plan of treatment to be adopted in each case.

In most instances, the causal treatment, even when combined with dietetic measures, is insufficient, and energetic *direct treatment* must be resorted to. A veritable chaos of remedies is known in practice for arresting convulsions, though in the majority of cases there is an entire absence of definite and clear indications for their use. Of late a disposition to limit the treatment to the employment of certain groups of curative agents has been observable, and these will here be briefly mentioned.

Electricity occupies a distinguished place amongst these, though the results of its action in these forms of neuroses are much less satisfactory than in others. It is possible that this is to be ascribed to inadequate and incorrect methods of application, since our knowledge of the localization of convulsive diseases is still very obscure. It will be sufficient here to give a few indications in regard to the electro-therapeutics of convulsive affections.

In the first place, the electric current is adapted for the treatment of the primary cause, since it has been shown to be effective in the treatment of diseases of the brain, spinal cord, or peripheral nerves, through its power of removing abnormal irritation of sensory nerves. On this depends the brilliant results that have been obtained by treatment with electricity, and especially with the galvanic current, which has, in many instances, proved of the greatest value. Very diverse modes of application of the current are occasionally effective, according to the nature and localization of the primary disease. For further details on this point the reader is referred to the various sections of this work, and to the manuals on electro-therapeutics.

The electric current also exhibits an antispastic action, which may become manifest in various ways: first, by lowering the excitability of the affected motor apparatus; secondly, by its action as a strong peripheric stimulus, in consequence of which it effects a reflex inhibition in the central organs, and thus suppresses spasm; and lastly, because the primary irritation being removed, the trophic and circulatory disturbances of the motor apparatus which maintained the convulsion are abolished; —all of which are effects of the so-called catalytic actions, especially of the galvanic current. Very various means are, it is well known, employed in electro-therapeutics to attain these objects. Experience shows that several methods must be often applied in succession before the most effective one is discovered. The faradic current may be applied with moist electrodes through the diseased nerves and muscles, with the object of lowering their excitability and exerting an alterative action; or it may be applied in the form of gradually increasing faradic currents (Frommhold), which, it would appear, lower the excitability still more energetically; or, lastly, strong irritation of sensory nerves may be effected by means of moist or dry electrodes, or the faradic brush, for the purpose of obtaining a centripetal spasm-subduing action. The galvanic current may also be applied in various ways. For instance, the persistent application of the anode, or of a descending current to the nerve and muscle, is said to lower their excitability; the transmission of a frequently interrupted simple galvanic current through nerve and muscle, is stated by Remak to exert a remarkable antispastic effect, which he has endeavored to explain by attributing centripetal action to it; more recently Benedict has especially recommended the application to the diseased nerve trunks of frequently repeated voltaic alternatives. Stabile currents, conducted through the supposed focus of the disease, are most effective in producing catalytic alterative actions. Ranke has observed that strong galvanic currents passed through the spinal cord suppress, by reflex inhibition, convulsions proceeding from it ; the direction of the stream is here of no importance, but it must be of sufficient strength.

This fact, which has been established in regard to the frog, appears also to have in certain cases some therapeutic value in man.

The electric treatment applied to the pressure points appears also to be quite worthy of attention, Remak in particular having obtained very remarkable results by this means. The treatment must vary according to whether it is applied to spasm-inhibiting or spasm-exciting pressure points ; and for this purpose the galvanic current is the more serviceable of the two.

It thus appears that the treatment of convulsions by means of electricity is extremely difficult. It is, in fact, almost impossible at present to give any precise indications for its use. There are indeed many methods of applying it, but we are too often reduced to empirical attempts to obtain its curative action. The results are consequently very uncertain, being in some instances both brilliant and persistent, whilst in other cases all methods entirely fail. Tact and knowledge on the part of the electro-therapeutist are here especially requisite.

The group of *narcotics* and *anæsthetics* supplies a series of agents which, however, are here of much less value than in neuralgia. It is only now and then that they act as palliatives, and only in rare cases that they prove really curative. Some of them are particularly effective in reflex convulsions, their action being rather to diminish the sensory excitability (as, for example, opium and its alkaloids, atropine, chloroform, ether, etc.), or to excite the cerebral centres which inhibit reflex movements (as has been demonstrated by Weil to be the action of digitalis); others again, like woorara,[1] diminish directly the excitability of the motor apparatus ; others, finally, like nitrite of amyl, digitalis, and bromide of potassium, exert a powerful action on the vasomotor nerves, and through them indirectly upon the spasms. No doubt can be entertained that all these actions may prove of value in different forms of convulsive diseases. Their use must, however, be directed according to the special indications of the particular case, and the details can only be given when the several forms are under discussion.

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[1] Weir Mitchell recommends the injection of atropine into the muscular tissue itself as being particularly effective in cramps and contractions.

The indications for the employment of the large group of *alteratives* and *nervines*, which include many powerful curative agents, are still less precise. These have for the most part been empirically discovered, and are also usually prescribed in an empirical manner. It is only in particular cases that indications for their use can be given. As a rule, they must be tried without any definite or certain motive. Energy and due persistency in prescribing them are the chief conditions for obtaining a successful result. Amongst these remedies we may mention the following, which in part also fulfil causal indications: iodide and bromide of potassium, arsenic (which, as Eulenburg has recently shown, constitutes an excellent remedy when applied subcutaneously in many forms of tremor), the preparations of zinc (especially the valerianate of zinc, which I have often found to be serviceable, and which appears to be especially useful in the later stages), nitrate of silver, hydrico-nitrate of bismuth, corrosive sublimate, valerian, artemisia, assafœtida, etc. There is scarcely a single remedy in the materia medica which has not at some time or another been recommended for convulsions (see the bibliography of the article on Epilepsy), a certain indication that our treatment of convulsions is not very successful.

Derivatives have of course been tried, and very definite results have been obtained with them; and even if we disregard the cases in which they fulfil the causal indication and cure the primary lesion, it cannot be denied that they have a really beneficial action in many forms of convulsion. This becomes intelligible if we consider the inhibitory influence on reflex actions which is possessed by the greater number of strong centripetal excitations; it may also perhaps be explained by the fact that powerful cutaneous irritations or inflammations act directly as alteratives upon the central nervous system, and induce changes in its circulation and trophic conditions, or in those of the adjoining parts. Hence in what appears to be appropriate cases, sinapisms, vesicants, moxæ, and especially the actual cautery, may be tried.

Busch[1] has very recently obtained such remarkably favorable results from the actual cautery in very chronic and obstinate cases affecting the muscles of the neck, throat, and arms, that it appears deserving of further trial. With a red-hot iron he burned the skin in band-like stripes, from three to five inches in length, on each side of the vertebral column, maintained suppuration for a little while, and kept the patient in the meantime in bed.

From a resort to *baths*, undertaken with a view of effecting directly a cure of the convulsive affection, little in general is to be expected. For the most part the best effects are obtained from indifferent thermal baths, with a relatively low temperature. On the other hand, baths often play an important part in fulfilling the causal indications, and at any rate they constitute one means of strengthening the tone of the nervous system. The same may be said of *hydrotherapeutical treatment.*

Gymnastics have, as a rule, but little value in the treatment of convulsion; in some cases, however, they may no doubt act advantageously by strengthening the influence of the will, and thus aiding in the removal of contractures and abnormal positions of the joints; a direct curative influence, however, can only very rarely be anticipated from their use. The necessary proceedings are described under the heads of the several forms.

Lastly, the *special surgical treatment* of convulsions is limited to a very few groups of cases. Certain forms of contractures are almost the only ones that are accessible to this method of treatment, and this is often extremely successful when tenotomy and orthopædic measures are judiciously combined.

On the other hand, tenotomy has for the most part proved futile in many tonic and clonic spasms of particular muscles and groups of muscles, and has therefore come of late years to be almost entirely neglected. The improvement is usually only

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[1] Berlin Klin. Wochenschrift, 1873. Nos. 37–39.

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transitory, and when the divided ends have again united, the spasm recurs. Section of the motor nerves implicated in the convulsive affection must not be risked without due consideration, on account of the unavoidable and almost incurable paralysis that is produced.

On the other hand, excellent results have been obtained in many cases by section of those sensory nerves in which a reflex connection with the convulsion can be demonstrated by pressure (as for example section of the supraorbital nerve in blepharospasm). According to von Graefe's observations, section of the nerves corresponding to the principal or primary pressure points is especially effective, whilst section performed at the so-called induced pressure points is only partially successful. Where it is possible the operation should be performed subcutaneously, and is, for the most part, free from danger; it has been already successfully undertaken in the sensory nerves of almost all parts of the body. The mode of action of this operation may, perhaps, be explained from the physiological facts given above, in speaking of the subject of pressure points. At the same time, it must not be unconditionally accepted that the effect of the operation is always the removal of peripheric reflex irritation, but we are in many cases compelled to admit that the operative procedure exerts such a centripetal action (reflex inhibition? alterative influence?) on the central nervous system that the cure of the convulsion speedily follows, just as we are compelled to admit that the same action results from neurotomy in many forms of neuralgia.

Particular Forms of Spasms.

Under this head we have to describe the spasms of particular muscles, or those occurring in the region of distribution of particular motor nerves and groups of nerves, as more or less independent affections. The muscles supplied by cerebral motor nerves, and by those arising from the cervical portion of the spinal cord, are most liable to such spasms; and we must here also mention certain forms of spasm which have been observed, more or less widely spread in various peripheric nerve territories, the central origin and anatomical cause of which have not been satisfactorily established. The spasms of the external and internal muscles of the eye producing lagophthalmus and strabismus spasticus, nystagmus, myosis, spasm of accommodation and hippus, must here be excluded from consideration as affections belonging to a special department. They either belong to the department of ophthalmic surgery, or they constitute subordinate symptoms of various diseases of the brain and spinal cord, or of forms of general convulsion, as epilepsy and chorea ; in the latter case they will be considered under the heads of these diseases. Little is known in regard to isolated and independent diseases of these muscles.

a. *Spasm in the Region of Distribution of the Trigeminal Nerve.—Masticatory Facial Spasm.—Trismus.*

Romberg, l. c.—*A. Eulenburg*, l. c.—*Rosenthal*, Nervenkrankheiten, l. c.—*C. Bell*, Physiological and Pathological Investigations regarding the Nervous System (Deutsch von Romberg), p. 85, p. 288. etc. of German edition.—*Speyer*, Trismus periodicus. Deutsch. Klin. 1853, No. 29.—*Germain*, de la contracture du masseter. Gaz. hébdom. 1863, No. 7.—*Remak*, über dentale Neurosen des Herzens. Berl. klin. Woch. 1865, No. 25.—*Leube*, Krampf. d. Muscul. pteryg. d. recht. Seite u s. w. Arch. f. klin. Med. VI., p. 273, 1869.

Under this heading is included spasm in the region of distribution of the motor portion of the fifth nerve, that is, in the region of the muscles of mastication, including the masseters, the temporals and the pterygoids. This form of spasms is usually bilateral, occurring but very rarely on one side alone.

Symptoms.—The spasms may either be tonic or clonic.

Tonic contraction of the muscles of mastication causes the lower jaw to be so drawn up that the two rows of teeth are powerfully pressed together, constituting the condition termed *trismus*, or *lock-jaw.* It is impossible to open the mouth either voluntarily or forcibly. The teeth can be only very little, if at all, separated, so that the introduction of food is often extremely difficult, and can only be accomplished through spaces between the teeth, or by other means, as by injections. Mastication is, of course, quite impossible ; the muscles of the jaw feel tense, often as hard as wood, and their outlines are brought into strong relief. Sometimes, though not always, acute pain is experienced in the muscles. If both sides are equally affected, the rows of teeth are opposed to one another in a vertical direction; on the other hand, the lower jaw may be somewhat protruded, or retracted, according to the particular muscles affected. In unilateral spasm of the pterygoids (as in the case recorded by v. Leube), the lower jaw may be thrust to the opposite side, the lower row of teeth projecting on this side beyond the upper.

When the cramp of the masticatory muscles is *clonic*, the lower jaw is moved backwards and forwards more or less regularly and energetically, either in a vertical or horizontal direction. In the former case chattering of the teeth occurs as in the rigors of fever ; in the latter case grinding of the teeth occurs —the sign which is so ominous in many diseases of the brain. True masticatory movements may be performed in a convulsive manner, as occurs in many epileptic, hysterical, and other forms of convulsions.

Little is known in regard to the concomitant symptoms that are so closely connected with convulsive diseases. On the other hand, phenomena are frequently observed which depend on the primary disease, such as neuralgic pains in the trigeminus, toothache, symptoms of periostitis in the lower jaw, cerebral symptoms, etc. Many disturbances may arise in consequence of this form of spasm: biting of the tongue and lips (Speyer), ulcerations and inflammations of the gums and oral mucous membrane, and conditions of inanition consequent upon insufficient supply of nourishment.

Etiology.—Conditions of excitation of the nerves themselves, such as inflammation, softening, development of tumors, and the like, rarely constitute causes of spasm of the muscles of mastication, which is for the most part unilateral. Catching cold is much more frequently the cause of trismus, which may also however occur quite independently, without being a symptom of general tetanus. Clonic spasms are observed most frequently in the cold stage of fever, and are then undoubtedly of central origin. The same may be said of those forms which accompany various forms of central disease, such as meningitis, apoplexy, cerebral tumors, local softening, etc., in which cases spasm of the muscles of mastication not unfrequently occurs as a perfectly isolated symptom. This form of spasm is often a concomitant symptom of certain forms of general cramps, as, for example, of epilepsy and eclampsia, of hysteria and chorea, and especially of tetanus, in which trismus is an almost constant, and for the most part initial symptom. (See Tetanus).

The isolated forms of spasm of the muscles of mastication most frequently result from reflex influence, acting through various nerves, though chiefly of course through the third branch of the fifth pair; the different forms of dental irritation, such as abnormal and difficult dentition, eruption of the wisdom teeth, caries and inflammation of the teeth, injury inflicted upon the lower jaw through extraction of the teeth, toothache, etc., being especially fruitful sources of spasm of the muscles of mastication. In addition, periostitis of the lower jaw, disease of the temporo-maxillary articulation, and occasionally also neuralgia of the fifth have been observed to constitute causes of these forms of reflex spasm. Disease of more distant parts, such as abscess in the groin, injury to the thumb, the presence of worms in the intestine, have all been observed to produce similar spasms, and there can be no doubt that in these cases some trophic disturbance must have been present in the central organs.

The *diagnosis* of these forms of spasm is very easy. The clonic form cannot indeed be confounded with any other, and in the tonic form, it is only necessary to avoid mistaking it for anchylosis of the lower jaw. A little care will easily prevent this mistake, since the nature of the joint and the position of the muscles of mastication, which are so readily accessible to external examination, greatly facilitates the differential diagnosis. In doubtful cases chloroform may be employed.

The *prognosis* essentially depends on the cause of the disease. As a rule, it is favorable in the rheumatic, idiopathic, and reflex forms; on the other hand, it is doubtful, and often unfavorable in diseases of the central nervous system, in severe general convulsions, and especially in tetanus.

Treatment.—In the greater number of cases the treatment must be essentially directed to the removal of the cause, as in diseases of the central nervous system, and in general forms of convulsion. In rheumatic cases active diaphoresis, derivation to the skin, and the administration of the iodide of potassium should be tried. In the reflex forms the origin of the affection should be discovered and the treatment directed to its removal. With this in view, a careful examination of the teeth and lower jaw should be made, and appropriate local treatment, such as the extraction of carious teeth, the application of leeches, incisions, the employment of galvanism should be adopted.

In reference to direct treatment, electricity must first be tried; and the above-mentioned methods of applying both the galvanic and the faradic currents (and especially the faradic brush) may be used with every prospect of success. In the next place, cutaneous irritants, such as mustard poultices, blisters, or even moxæ applied to the neck may be used, and may be advantageously combined with the internal use of narcotics, such as opium and morphine, cannabis indica, and atropine; subcutaneous injections of morphia often prove very effective. In obstinate cases resort must be had to specifics, such as arsenic, iodide of potassium, valerianate of zinc, etc. In chronic cases, an attempt has sometimes been made to effect gradual separation of the jaws by mechanical means, as by the daily introduction of progressively thicker wedges of wood between the teeth.

In serious cases particular attention should be paid to the food of the patient. It is often requisite to give the nourishment in a fluid, and as far as possible concentrated form (such as eggs in milk, extract of meat, strong broths, etc.), which may be administered by means of a tube introduced through a space in the teeth, or through the nose, or by the employment of nutritive injections of mixtures of pancreatic juice and meat, milk, soups, wine, etc.

b. *Spasm of the Muscles supplied by the Facial Nerve.—Spasmus Facialis.—Mimetic Spasms of the face.—Convulsive Tic.*

Text-books of *Romberg*, *Hasse*, *A. Eulenburg*, and *M. Rosenthal.—Benedict*, Nervenpath. und Electrotherapie, 1874.—*C. Bell*, l. c., p. 243 et seq. (of the German translation).—*François*, Essai sur les convuls. idiopath. de la face. Brux. 1843. —*Cullerier*, Contracture des muscles de la face. Gaz. des hôp. 1853, No. 31.—*A. Leineweber*, de spasmo faciali. Diss. Berolin., 1858.—*Oppolzer*, Allg. Wien. med. Zeit. 1861 No. 10.—*A. von Graefe*, Arch. f. Ophthalm. I. 1. p., 440; IV. 2. p. 184; IX. 2. p. 73. Deutsch. Klin. 1864, No. 20 u. 24, 1865, No. 27.—*Remak*, über Gesichtsmuskelkrampf. Berl. klin. Woch. 1864, Nos. 21–23; 1865 No. 27.—*Debrou*, sur le Tic non douloureux de la face. Arch. génér. Juin 1864. —*W. Erb.* Krampf des Facialis. Arch. f. klin. Med. V., p. 518. 1869.—*Deleviéleuse*, Tic facial; section des nerfs. Guérison partielle. Gaz. méd. de Strasb. 1869, No. 6.—*Seeligmüller*, über intermitt. Blepharospasmus. Zehender's klin. Monatsbl. IX. 1871.

Under these terms are included cases of spasm of all the muscles supplied by the facial nerve; these are among the most frequent of all forms of convulsion, which is, to some extent, intelligible when we consider how frequently the muscles supplied by the facial nerve participate in various forms of movement (as in speaking, in expression, in mastication, and in respiration), and also how numerous are the direct and reflex excitations, which constantly affect this region. All degrees of spasm are met with, from physiological contraction to abnormal spasm. Under the influence of the emotions and of sudden fits of passion, the movements of the face ministering to expression not unfrequently attain in excitable individuals such a height, that they cannot easily be distinguished from actual spasm, and when such movements become fixed and habitual, they already represent true spasm. All such movements, the degree and persistence of which surpass physiological limits, must be classed among the spasms; the same is true of all muscular contractions depending on purely pathological excitations. We shall here consider exclusively the more or less independent spasms occurring in the region of distribution of the facial nerve, whilst those contractions of the facial muscles which constitute concomitant symptoms of more general forms of convulsion will be left for consideration in other chapters of this work.

Spasms of the facial muscles may occur on one or on both sides; they may affect a single muscle or all those supplied by the facial, in consequence of which they have been divided into the *partial* and the *diffused* forms, and it is advantageous to consider them under these headings.

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1. *Diffused Spasm of the Facial Muscles, True Convulsive Tic.*—This represents the characteristic form of spasm of the muscles of expression, and occurs especially in the clonic form, which is by far the most frequent and important.

In such cases a very remarkable type of disease is presented. There is a continually alternating contraction of nearly all the facial muscles supplied by the portio dura, a series of spasms which cannot be very exactly described. There is an unceasing and unmeaning contraction of the facial muscles, causing grimaces, which are the more striking when, as in most unilateral cases, the other half of the face is at absolute rest. Winking of the eyes, wrinkling of the forehead, contractions of the muscles of the nose, drawing up of the angle of the mouth, movements resembling those of laughter, follow in quick succession, or appear once or twice as clonic spasms; in the intervening periods, a few tonic and more persistent contractions of particular muscles are observed; finally, the whole expression of the face becomes quieter, a few twitches occur, and there follows then a longer or shorter pause.

In these attacks the muscles are not all equally affected in different instances; generally the spasms are confined chiefly to a few muscles, such as the orbicularis palpebrarum, the levator labii superioris alæque nasi, the zygomatici, corrugator supercilii, and more rarely the frontalis or the platysma, and still less frequently the muscles of the ear. I do not find that in any instance movements of the velum palati have been observed during the spasm, nor have I been able to establish the occurrence of such movements in the cases that have fallen under my notice.

Facial spasms occur in *paroxysms* of variable duration, sometimes lasting only a few seconds, and more rarely as long as one or two minutes. They commence with a few twitches, which gradually increase in energy and rapidity, ultimately lead to one or two tonic contractions, and then gradually again subside. In the intervals all is at rest, or only a few slight contractions occur, which are associated with any reflex or voluntary movements that the patient may happen to make. The frequency with which the paroxysms occur varies greatly; in some instances only from three to ten occur in twenty-four hours, whilst in other instances, they follow each other so rapidly that there are from twenty to forty attacks in an hour. The attacks do not always cease, even during the night, though they are then less frequent (Oppolzer.) They often come on without any obvious cause, though they are often also produced by definite causes, as by speaking, voluntary movements, closure of the eyes, strong impressions of light, loud sounds, sensory irritations of the face, the emotions of anger, grief, terror, etc.

The spasms do not always occur in a well-marked attack, but there are cases in which twitchings are observed here and there, that only undergo variations in their intensity and distribution; slighter cases are also met with, in which the spasms appear to be connected only with definite external or internal causes (irritation of the skin, voluntary excitations), and never attain a high degree of development. This occurs in those forms of spasm which so frequently occur as a consequence of well-marked paralysis of the facial nerve.

Notwithstanding the apparent severity of the disease, the influence of the will upon the affected muscles is not materially impaired; the free action of the facial muscles is of course impossible during the paroxysm itself, but the patients are quite able to perform all voluntary movements in the intervals, and the movements of expression do not then appear to be disturbed.

In many instances an extension of the convulsion is observed to take place to adjoining muscular regions; the muscles of mastication and those of the tongue are most frequently thus affected. How far this occurs in the case of the muscles of the eye, the implication of which in certain cases, according to the recent physiological investigations of Hitzig, might be expected, we are ignorant, since the spasmodic closure of the lids, which is usually present, renders it difficult to observe them. At the height of the attack, the muscles of the neck and shoulders, and even of the arms, are often spasmodically affected, and an extension of the convulsion to the originally unaffected half of the face may often be observed. In this form of spasm all sensory phenomena, and especially pain, are so constantly absent that in contrast to tic douloureux it is often termed "indolent tic," and when some authors refer to the fearful pain accompanying spasm of the muscles of expression, it is obviously consequent on their confounding the two diseases. This mistake may, undoubtedly, easily be made in those cases of tic douloureux which are accompanied by well marked reflex spasm of the facial muscles, and which present the characters of convulsive tic. Careful observation will, however, easily show that in such cases a primary neuralgia of the trigeminus is present, and that severe pain is not produced by the contractions of the facial muscles. It is evident that these and other sensory disturbances (anæsthesia, formication, etc.), which sometimes accompany spasms of the muscles of expression, are to a greater or less degree symptoms of an accidental nature.

Vaso-motor and *trophic phenomena* have scarcely ever been reported amongst the concomitant symptoms of this form of spasm ; at all events, they never constitute prominent features. Since the view that the facial nerve contains no vaso-motor or trophic fibres may now be regarded as erroneous, the conclusion is admissible that the pathological conditions leading to facial spasm usually attack the nerve at some spot where no vaso-motor fibres exist. The reports of the disease are also silent in regard to *disturbances of secretion*, although one would be disposed to expect changes in the salivary secretion. Bouvin mentions a case in which a remarkable saline taste was perceived in the mouth. One of my patients perceived a roaring in the ear during the attack, and in another case a ringing in the ear was experienced during the contractions (spasmodic contraction of the stapedius muscle ?).

The *electrical excitability* of the branches of the facial nerve, and of the muscles of the face, undergo no remarkable alteration. I have, indeed, in some cases found it to be both quantitatively and qualitatively perfectly normal.

Pressure points are only discoverable in a minority of the cases in this diffused and clonic form of facial spasm ; they are more frequent in the cases limited to the orbicularis palpebrarum, or in those of partial spasm of this muscle (blepharospasm) ; but inasmuch as this may pass into the diffused form of spasm of the facial muscles, pressure points may also be present in the latter condition. They occur in every branch of the trigeminus, in the skin of the face, and in the cavity of the mouth, and may be divided into the already mentioned categories of primary and induced pressure points. Remak has also called attention to certain points in the cervical region of the spinal column (transverse processes that are situated at the level of the different cervical ganglia of the sympathetic) on which, when pressure is made, or to which, if galvanic treatment be applied, arrest of the spasm and a cure may be effected. He associates these with sympathetic nerve fibres distributed to the arteries at the base of the brain, and attributes the curative effects of the galvanic current to its indirect catalytic action. Similar inhibitory pressure points effecting arrest of the spasm may sometimes be found in more remote parts of the body, as over the brachial plexus, the vertebral column, and the wrist, and no pains should be spared to discover them.

Tonic spasm of the facial muscles presents essentially different features. The half of the face affected exhibits a peculiar rigidity; it remains at rest during mimetic movements; the furrows are deeper; the face is slightly drawn towards the affected side; the angle of the mouth is distorted; the fissure of the lid is smaller, and the eyebrow is drawn upwards; mastication is rendered difficult upon the affected side, in consequence of the deficient mobility of the cheeks; a disagreeable sensation of tension is experienced in the muscles affected, the fixed and stiffened condition of which is only interrupted occasionally by sudden twitchings of one or several muscles. Trismus of the same side often accompanies this tonic spasm of the facial muscles. If the spasm affect both sides, a peculiar and characteristic aspect is presented by the face, which König has described as constituting a striking symptom in tetanus.

The reports published on this form of mimetic spasm of the face are comparatively few in number. I entertain no doubt, however, that most of these cases are only those forms of muscular contracture which so commonly occur in the later stages of severe facial paralysis, and with which clonic contractions of the facial muscles are subsequently so often associated. A few cases, nevertheless, are due to other and unknown causes.

The *etiology* of spasm of the facial muscles has only in a few cases been satisfactorily ascertained.

The *predisposition* to it is also not very common, since upon the whole it is a rare form of neurosis. Men are somewhat more frequently affected by it than women. Those who have a neuropathic diathesis are particularly liable, and it is not unfrequently hereditary. Anæmia, chlorosis and hysteria predispose to it.

Catching cold is one of the most frequent direct causes. There are many examples on record of this, and it is the usual cause in those who are otherwise healthy and robust; its mode of action is altogether unknown. *Reflex irritation* is still more important as a cause of the disease, especially such as proceeds from the fifth pair of nerves. Thus, on the one hand, affections of the eyes, such as various forms of inflammation, keratitis, ulceration, wounds, foreign bodies, ciliary neuralgia, etc., and, on the other hand, affections of the jaws and teeth, caries of the teeth, ulcerations in the mouth, periostitis, parulis, etc., are the most frequent exciting causes of such spasms, which then for the most part begin as partial spasms, and gradually, in predisposed persons, or, in cases where the irritation is continuous, become diffused spasms. Injuries of the face and temples, as well as trigeminal neuralgiæ, have also been observed to constitute causes of the affection, and it is especially those severe so-called convulsive forms of trigeminal neuralgia, particularly described by Trousseau as *tic douloureux*, which often present an exquisite picture of mimetic facial spasm. Reflex convulsive tic has often also been observed as a consequence of irritation of more remote nerve regions, as in cases of uterine disease, worms in the intestinal canal, etc. According to Remak, cervico-brachial neuritis is often a point of origin for this form of spasms.

Direct Irritation of the Facial Nerve in its peripheric course at the base of the brain, or in the temporal bone, is rarely the cause of the disease; occasionally, however, it has been observed as a result of tumors at the base of the skull, and in caries of the temporal bone; in such cases it is important to notice carefully whether the contractions are tonic or clonic.

Irritation of the facial in its central course, and of the facial nucleus, in the anterior lobe of the cerebrum itself, is not unfrequently the cause of spasms of the facial muscles in cerebral diseases, as is sufficiently demonstrated in cases of abscess of the brain, cerebral tumor, and apoplexy. The interesting observations of Wernher and Hitzig, as well as case 5 of those reported by Debrou, are especially important in this point of view, and show that, with pathological conditions of irritation near the "facial nucleus," clonic spasms occur in the muscles supplied by the facial nerve. The question arises whether many cases of the so-called idiopathic spasm of the facial muscles do not arise from palpable or impalpable disease of this facial nucleus, a question the reply to which is of the greatest importance in determining the point to which therapeutic agents should be applied.

Facial spasms caused by *psychical conditions*, such as anger, fear, and other mental stimuli, may perhaps be associated with the foregoing. On the other hand, the facial contractions that occur in consequence of severe facial paralyses, are perhaps referable to abnormal excitability in the reflex organ of the facial situated in the medulla oblongata, as Hitzig has endeavored to demonstrate: whilst the contractures of the facial muscles, that very commonly occur after such paralyses, possess only a muscular origin.

Mimetic facial spasms often develop without any obvious cause, and, after rising to a certain degree of intensity, become habitual.

Lastly, it is here only necessary to mention that facial spasm not unfrequently occurs as a secondary symptom of the various forms of general spasms, as in epilepsy, eclampsia, chorea, tetanus, etc.

The *course* of the disease is extremely uncertain and irregular, sometimes steadily improving or progressing in intensity, sometimes presenting many remissions and exacerbations. Relapses are very common. The duration of the disease is very variable; occasionally, but rarely, it lasts only a few days or weeks: generally, however, it is extremely chronic, persisting for months or years, and continuing, in some cases, even throughout life. In regard to the ultimate issue of the disease, recovery is unusual, whilst partial recovery with strong disposition to relapses is more common. Not unfrequently the affection becomes habitual and persistent, while the primary disease may of course often lead to still worse results.

The *diagnosis* of this form of spasms offers few difficulties in practice, since the disturbances which characterize it are of a very remarkable nature; it is only in cases of tonic contraction of the facial muscles that care must be taken to avoid mistaking it for facial paralysis, which may easily be avoided by a little attention. It is much more important and more difficult to determine the seat and the true cause of the affection, since even with the most careful investigation and the greatest acuteness, it is not always easy to ascertain whether the disease is due to peripheral irritation or to disease of the central nervous system; whether it has its origin in reflex excitation, and if so, what is the position of that irritation; it is unnecessary to pursue this subject in further detail.

The prognosis is sufficiently clear from what has been said in regard to the causes and the course of the disease; it is only distinctly favorable in those cases where there is a distinct and easily removable reflex irritation; in most cases it is doubtful, and in some forms, especially in those that have become chronic, it is decidedly unfavorable; even in these cases, however, a perfect cure is sometimes obtained (Remak). The nature of the primary disease of course influences materially the prognosis.

The *treatment* of mimetic facial spasms is one of the most thankless problems of medical practice. However slight and free from danger the disease may appear, it still often bids defiance to all conceivable remedial measures.

The first therapeutic means to be adopted must obviously be those which may effect the removal of the cause of the disease. In recent rheumatic cases energetic diaphoretics, vapor baths, and derivatives may be tried; but where the spasm is reflex, an attempt should obviously first be made to remove the sensory irritation by treatment of any present ophthalmic affection, by the extraction of teeth, by the cure of ulcers, etc. Neurotomy of the affected sensory nerves, especially of the supra-orbital, may be undertaken, and brilliant results have often been obtained by this means. If it be a symptomatic affection, the cerebral lesion must be treated.

Electricity is at present the best direct means of treatment, but I am unable to agree with Benedict that it has a favorable action in recent cases, and an unfavorable one in chronic ones, for I have in many quite recent cases tried without effect all conceivable electro-therapeutic means, and it has often appeared to me that a certain duration of the disease is favorable for the action of electricity. The information we at present possess is insufficient to determine this point, but on the other hand, the numerous favorable accounts that have been reported render it expedient to employ electricity, though perhaps in different ways in different cases. The following methods of application may be tried: the anode may be kept applied (stabile), with an increasing and then diminishing strength of current, on the plexus anserinus or behind the ear; a descending stabile galvanic current may be passed from the nerve to the muscles; voltaic alternatives may be applied to the several branches of the nerve (Benedict); galvanic currents may be passed transversely through the mastoid processes, and perhaps also direct treatment may be applied in the neighborhood of the facial nucleus in the parietal region; in forms dependent on central disease, galvanization of the sympathetic nerve in the neck may be practised, with special regard to those ganglia which modify the spasm when pressure is applied to them; galvanic treatment (the anode being used) may in appropriate cases be applied with good results to the pressure points (Remak); lastly, progressively increasing faradic currents may be applied to the nerve with special benefit (Frommhold). All these methods should be applied with energy and perseverance, and even then they often fail.

The subcutaneous injection of morphia is one of the most important means of applying *narcotics;* it should be introduced in the vicinity of any pressure points that may be present, or into the skin of the temples. Good results have been obtained by means of injections of woorara, atropine and strychnine, and also from the inhalation of chloroform, or its application on wool to the plexus anserinus.

It is often necessary to employ *nervines*, such as arsenic, oxide and valerianate of zinc, nitrate of silver, assafœtida, valerian, etc. All that was said in regard to them under the head of the general treatment of spasms applies here, but it is impossible to give any precise indications for their use.

Strong exertion of the will appears to be occasionally effective in curing the disease, as is shown by a case recorded by von Graefe, in which a girl was cured of her blepharospasm by ordering her to get up on and down from a chair several times in succession, a procedure which made it necessary for her to open her spasmodically closed eyes.

The *neurotomy of sensory nerves*, especially of the supra-orbital, often appears to be followed by good results, even where no reflex irritation proceeds from those nerves (Romberg, von Graefe), and this operation being comparatively harmless, may be performed in doubtful cases, even though it often proves unsuccessful. Is it possible that its occasional success is referable to centripetal action, and can be placed in the same category with strong irritants applied to the skin?

Section of the facial nerve itself has only been ventured upon in a few cases, on account of the unavoidable paralysis that succeeds the operation. Delevieleuse has performed it on both sides, though only with partial success. The motility returned—so it is reported,—soon after the operation (?). Firm pressure upon the trunk of the facial must be applied with great caution.

Dieffenbach, in a very obstinate case, divided subcutaneously most of the muscles affected with cramp, and thus obtained a very favorable result.

2. *Spasms of Isolated Facial Muscles.*

Isolated muscles of the face on one or both sides are not unfrequently affected by spasm, though it must also be admitted that in diffuse facial spasm particular muscles only may contract at any given moment. It is characteristic of partial spasm that it is always limited to the same muscles; thus, for example, partial contraction may occur from time to time in the zygomatici and the adjoining muscles, causing the face of the patient to be distorted with convulsive smiles (*risus sardonicus*); or spasmodic contraction may take place at intervals in the corrugator supercilii, or the alæ nasi and upper lip of one side may tremble convulsively, or the ears may exhibit lively and unwonted movements in consequence of spasmodic contractions with or without coincident contractions of the occipito-frontalis (Romberg, Hoppe). Partial cramps limited to muscles surrounding the eyes are by far the commonest of these affections, and we must devote a short section to a description of these "palpebral spasms," since they are of great practical importance both on account of their frequency and on account of the remarkable distortions which they produce.

A large number of very remarkable cases of *tonic spasm of the lids* (*blepharospasm*) have been recorded, and we are particularly indebted to von Graefe for great advances in our knowledge of its pathology and treatment.

By this term is understood a tonic contraction of the orbicularis palpebrarum, appearing in paroxysms of variable duration. The closure of the lids may be either intermittent or persistent, the several attacks may last for a few minutes or for hours, or they may even extend over weeks and months, so that the patient may be regarded as completely blind. No description can exhaust the bizarre forms of contraction often presented by this affection, for the sudden closure of the eyes is accompanied by many grimaces, caused by contraction of the antagonistic muscles, which attempt in vain to remove the compulsory blindness by opening the lids. The attacks may be induced by many circumstances, the most frequent being the action of light, straining of the eyes, firm closure of the lids or the attempts to open them forcibly, loud sounds and mental excitation. They often supervene quite suddenly, the lids becoming suddenly closed, and remaining in that condition for a variable period; they not unfrequently open again with equal suddenness when pressure is made on definite points, the lids separating as though by action of a secret spring.

It is in this form of spasm that the *pressure points* which are capable of inhibiting spasm have been recognized and studied. They are often easily and at once discovered, and in some instances the patient has even found them out himself, and has employed them for the instantaneous removal of the spasm. In other instances they are only to be discovered after tedious and protracted searching, and are then sometimes found in quite unexpected parts of the body. Strong pressure exerted upon these points speedily removes the spasm, and, in some instances, quite suddenly, so that the lids open as by a spring. For the most part the inhibitory action of the pressure on the spasm continues for some time; less frequently it only lasts as long as the pressure is applied, the contraction returning as soon as it is removed. Such pressure points are of course found most frequently in the region of distribution of the trigeminus, and they must be distinguished from the well-known "painful points" of Valleix, which are present in neuralgia; such a pressure point is often situated at the foramen supra-orbitale, but they have also been found along other branches of the trigeminus, as on the skin of the face, the gums, the fauces, the malar bone, and the mastoid process. If they are not found at these points they may be sought for over the spines and transverse processes of the cervical vertebræ, over the sympathetic, over the brachial plexus, in the wrist, etc. In many instances there are several or even numerous pressure points, whilst in others they are completely absent. In the latter case arrest of the contraction may be effected by the action of the galvanic current at definite points (Remak). The distinction between primary and induced, or secondary, pressure points, which has been established by von Graefe, should be borne in mind.

It need scarcely be remarked that this form of spasm is extremely annoying to the patient, the immediate consequence of the closure of the lids being the periodic or persistent abolition of the power of vision. Coincidently with this there is a high degree of photophobia, pain in the eye and forehead, and the general features of the primary ophthalmic affection. Pain as well as all kinds of nervous disturbance in very various parts of the body may be experienced.

In the larger number of cases blepharospasm is of reflex origin, and the fibres of the fifth pair distributed to the eye appear to be the chief conductors of the reflex irritation. Amongst the commonest causes are all the various inflammations of the eye, especially scrofulous, herpetic, and ulcerative inflammations of the conjunctiva and of the cornea, wounds of the eyes, the entrance of foreign bodies, photophobia, and supra-orbital neuralgia.

Blepharospasm has also been observed as a consequence of ulceration in the mouth and throat, of carious teeth, and of neuralgia of the different branches of the fifth, and the existence of pressure points in remote parts of the body seems to show that blepharospasm may be arrested in a reflex manner through other nerves. That the spasm is in such cases almost always either originally bilateral or that it becomes so in course of time, is attributable, apart from the law of reflex symmetry, to the commonly associated physiological action (as well as to the preformed anatomical connection ?) of the two orbiculares.

In the *treatment* of this form of spasm attention to the causal indications is of the utmost importance, and the greater number of the cases recover as soon as the inflammation of the eye, or the trigeminal neuralgia which has produced them, is removed, or after the extraction of foreign bodies ; at the same time, the study of the pressure points is of much importance in the treatment of blepharospasm, and extraordinary cures have been obtained both by neurotomy of the affected nerve trunks (von Graefe), and by the action of the galvanic current (stabile anode, Remak). Neurotomy of the supra-orbital nerve has also been followed by brilliant results in this form of spasm, and a strong indication for the operation is afforded by the removal of the spasm when pressure is made upon the supra-orbital foramen ; it should not, however, be undertaken till other means have been tried and have proved ineffective.

Amongst other measures, the subcutaneous injection of morphia, which should be performed either in the neighborhood of the pressure points, or in the temples, deserves mention. Atropine and belladonna also constitute good palliatives. though usually the beneficial effects are not experienced until manifestations of belladonna narcosis appear.

Galvanic treatment should be especially applied to the pressure points, but may also, in appropriate cases, be applied to the eye itself, or to the supra-orbital nerve ; and the remarkable results obtained by Remak from galvanization of the cervical portion of the sympathetic nerve, should be borne in mind. The methods described above, as applicable to convulsive tic, may also be practised.

Von Graefe strongly recommends the methodic plunging of the face into cold water, for the cure of the blepharospasm which so often follows keratitis in children. Cold or warm compresses, narcotic fomentations and cataplasms to the eye may occasionally prove beneficial. Little good can be anticipated from pressure methodically applied to the facial, at the stylo-mastoid foramen, and Sir Charles Bell has strongly protested against section of the facial nerve; at any rate, the operation should only be resorted to in the most extreme cases.

Clonic Contraction of the Lids—Spasmus Nictitans–Nictitatio—differs only in form, and but little in its nature, from tonic contraction of the eyelids. It consists in a convulsive blinking, or alternate closure and opening of the eyelids, caused by clonic spasm of the orbicularis palpebrarum, in which other neighboring muscles (as the corrugator supercilii, occipito-frontalis, and zygomatici) more or less extensively participate; many men exhibit them in a slight degree when any unusual stimulus, such as a bright light, the reflection of the sun, &c., strikes the eye, and when they are in a state of nervousness or are affected by any mental emotion. In the higher degrees this form of spasms becomes an extremely annoying affection. It may occur spontaneously, but is often associated with tonic contractions of the lids, so that the clonic contractions introduce and terminate the attacks of tonic convulsion.

This form of spasms is also in most instances of reflex origin, and is in this respect quite analogous to blepharospasm. It would be a mere repetition of what has already been said in regard to tonic contraction of the lids, were we to repeat in regard to nictitation what has been already stated concerning reflex irritation, pressure points, and the paroxysms in blepharospasm; why in some individuals a tonic contraction of the lids occurs, whilst in others, apparently exposed to similar conditions, the contractions of the lids are clonic, we are entirely ignorant; all that can be said upon the point is a pure matter of opinion.

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The *treatment* of spasmus nictitans must be conducted upon the same principles as those which guide us in the treatment of blepharospasm.

c. *Spasm of the Muscles Supplied by the Hypoglossal Nerve.—Lingual Spasm.*

Romberg, loc. cit. p. 387.

An independent isolated spasm of the muscles of the tongue supplied by the hypoglossal nerve is of extreme rarity, but on the other hand it is a very frequent concomitant symptom of various sensory and motor neuroses.

Lingual spasm appears as a restless, rolling movement of the tongue, or as a rapid protrusion and retraction of the organ, or as a convulsive protrusion ; in short, as a rapid and irregular succession of all the movements of which the tongue is capable. It may also appear as a tonic contraction of one or other muscular fasciculi, so that the tongue is kept for a variable period in some abnormal and bizarre position, or it may appear in the form of separate short contractions of the whole tongue, or lastly, in the form of fibrillar contractions of the lingual muscles.

Numerous cases have been reported which demonstrate that lingual spasm occurs occasionally in meningitis and other forms of cerebral disease (irritation of the hypoglossal centre in the anterior lobe of the cerebrum, or of the hypoglossal nucleus in the medulla oblongata?); that it is also a very common symptom in all forms of general convulsions (in hysterical convulsions, chorea, epilepsy, and eclampsia) ; that it not unfrequently accompanies trismus, spasm of the facial muscles and stuttering; that it may be a reflex symptom in trigeminal neuralgia, especially of the third branch of the fifth (Romberg), in caries of the teeth and ulceration of the gums (Mitchell), in progressive muscular atrophy and in bulbar paralysis, in which affection it commonly presents the form of fibrillar contractions. In regard to all these forms of lingual spasm, the more important points have been noted in the different sections of this work devoted to the description of the above-mentioned forms of disease. We do not possess at the present time sufficient information to enable us to give an account of the symptoms of independent lingual spasm or of its treatment, and it is very desirable that cases of the kind should be reported. I have recently seen a case in a girl, who suffered in addition from peculiar hallucinations, and in whom it occurred quite independently and in an isolated manner in the form of a frequent protrusion of the tongue and of an uncommonly rapid protrusion and retraction of the whole organ.

d. *Spasm of the Muscles Supplied by the Nervus Accessorius (Torticollis.—Tic Rotatoire.—Nickkrampf, etc.).*

See the treatises of *Romberg*, *Hasse*, *A. Eulenburg*, *M. Rosenthal*; *Benedict*, Nervenpathol. etc. 1874.—*Duchenne*, Electris. localisée. II. edit, 1861, p. 888 ff.—*Beard and Rockwell*, Medic. electricity, p. 461.—*Stromeyer*, Beitr. z. operat. Orthopäd. 1838, p. 128.—*Ebert*, das krampfh. Kopfnicken der Kinder. Annal. d. Charité, 1850. Bd. 1.—*G. Hirsch.* klin. Fragmente. I. 1857.—*Leconest*, Torticollis intermittent. Union méd. 1861 No. 67.—*Busch*, Anwendung des Glüheisens bei Krämpfen, etc. Berl. klin. Woch. 1873. No. 37—39, (Sitz. d. Niederrhein. Ges. in Bonn.)—For reports of cases, see *C. Bell*, l. c. pp. 342–359 (German translation).—*Schützenberger*, Gaz. méd. de Strasb. 1867, No. 9.—*Morgan*, Lancet, 1867, Aug. 3.—*Delstanche*, Presse méd. 1867, No. 15.—*Fournier*, Thèse de Strasb. 1870, etc.—*Stich*, D. Arch. f. klin. Med. Bd. XI. p. 524.

The not unfrequent cases of spasms included in this category implicate the sterno-cleido-mastoid and trapezius muscles. These muscles may be affected separately or both together; the spasm may be uni- or bilateral, and they may occur either in the clonic or in the tonic form, or they may assume the form of a persistent contracture of these muscles.

1. *Clonic Spasm of the Muscles Supplied by the Accessorius.*

With unilateral affection of the sterno-cleido-mastoid the head is moved convulsively backwards and to one side in a very characteristic manner (the chin is turned towards the opposite side and raised, the occiput is drawn backwards, the ear and mastoid process approximated to the clavicle of the same side: these movements, like those accompanying most of the forms of spasms that we shall now proceed to describe, may best be simulated by local faradisation of the muscles affected, and a precise study of the movements and positions that may be thus produced materially facilitates the diagnosis of such forms of local convulsions). This is a form of the *tic rotatoire* of the French, which, however, may also be produced by many other muscles which aid in the rotation of the head.

If the trapezius be alone spasmodically affected, the head is drawn backwards and towards the affected side without rotation of the chin; at the same time the shoulder is raised, the scapula is approximated to the vertebral column, and, according to what particular fasciculi of the muscle are chiefly affected, it is more or less rotated on its axis. The head is sometimes drawn backwards with great force, so that the occiput and shoulder almost touch.

If both the muscles of one side are coincidently affected, the two movements are combined, or alternate with one another; and the appearance presented will vary according to the preponderance of the action of one or the other muscle. In bilateral spasm of the muscles supplied by the accessorius, the head is, for the most part, drawn alternately from one to the other side; the chin is moved more or less quickly to one side or the other. More rarely a precisely synchronous contraction of the muscles of both sides is observed, which is in such cases usually limited to the mastoidei, in consequence of which an alternate elevation and depression of the chin (nodding) occurs with a more or less rapid *tempo*. To this belong the nodding movements, chiefly observed in children, in which the head makes, with greater or less rapidity, a great number of regular nodding movements. I think, however, that it is doubtful whether these movements are not chiefly caused by other cervical muscles.

All these contractions occur, for the most part, in *paroxysms* of variable duration, often lasting for a day, and not unfrequently coming on with such frightful vehemence that the head is tossed to and fro with great force, making the life of the patient miserable. In some instances the spasm is observed to be almost continuous; sleep, however, usually brings rest, though this is often prevented or delayed. Various circumstances induce the paroxysms, such as walking, talking, mental excitation, etc.; and their intensity is regularly increased when the patient is conscious of being observed, or is nervous.

At the height of the paroxysm, more or less numerous adjoining, or more remote, muscles are frequently affected; as, for instance, those of expression and mastication, those of the eye, shoulder and upper arm. In many instances the spasm of the muscles supplied by the accessorius is only a symptom of spasms affecting many groups of muscles.

Amongst the *sensory phenomena*, that which is most frequently mentioned is a painful state of exhaustion in the muscles affected with spasm. Pain, also, is not unfrequently complained of in the neck and occiput, in the shoulder and arm; and there is sometimes, also, formication in the skin of the arms. *Pressure points* are rarely present; if present, they are generally found in the trunk of the accessorius itself, in the brachial plexus, and in the vertebral column, but they may also be sought for in the affected muscles. In the higher degrees of the disease remarkable sequelæ are observed, amongst which disturbances of mastication, of speech, and of sleep may be specially mentioned. The depressing action of this tormenting affection on the psychical faculties is not to be under-estimated.

The disease usually develops quite gradually, the more serious forms rarely supervening suddenly; when moderate, the affection may long remain unchanged, and recovery may even take place. Usually, however, it increases in intensity until it attains an almost unbearable violence.

The *terminations* of this form of spasms may be manifold: recovery may take place, but this is a comparatively rare termination; or the spasms may become habitual, or they may pass into more or less severe forms of convulsion, such as epilepsy; or, lastly, they may terminate in paralysis. Which of these conditions shall occur depends on the nature of the cause.

The etiology of clonic spasms of the muscles supplied by the accessorius still remains, in many points, obscure. It can seldom be stated at what point of the accessory nerve, in its course through the spinal cord, medulla oblongata, or brain, the morbid stimulus acts; this only we know, from the cases that

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have been put on record, that the irritation producing the spasm may affect the nerve in very different parts of its course. Thus, this form of spasms has been observed in various diseases of the central nervous system (tumors of the brain and spinal cord, softening of these parts, meningitis, etc.); in injuries of the skull and neck; in diseases of the vertebral column in the cervical region (caries, periostitis, and tumors), in which case we must suppose a pathological irritation of the spinal cord in the cervical region, or of the roots of the accessory nerve to be present.

Catching cold is also a frequent cause, and may be brought about by sitting in draughts of air, by getting thoroughly wet whilst perspiring, and by other injurious influences of a similar kind.

We are obliged also to admit that these spasms may owe their origin to reflex irritation; in this category belong those cases which result from severe dentition, from worms, from disturbances of the digestion, and from uterine diseases.

To complete the consideration of this subject, it must be mentioned that spasm is a frequent consequence of emotional excitations, and that it occasionally makes its appearance in the puerperal state, and as a sequela of typhoid fever. We are still entirely ignorant of its pathogenesis in these cases.

The *diagnosis* of clonic spasm of the muscles supplied by the accessorius in well-marked and severe cases, in which the muscles at each contraction form distinct and prominent swellings, is not difficult. On the other hand, in those cases where the movements of the head are only slight, and scarcely observable, consisting only of a trembling or shaking motion, a very exact and careful examination is required for its recognition. In such cases all the already mentioned aids to diagnosis must be employed in establishing what muscles are affected with spasm. The determination of the true seat and cause of the disease is usually a much more difficult matter, and even when the cause of the spasm is external and well known, the exact localization and nature of the disease is often obscure.

The *prognosis* depends essentially on the nature of the primary lesion. In diseases of the central nervous system, and of the cervical vertebræ, it is of course very bad, and it is also unfavorable in idiopathic forms to which neuropathic individuals are particularly liable, as well as in habitual forms. On the other hand, it is rather favorable in the so-called rheumatic cases.

The *treatment* must be directed in accordance with the causal indications, and it is unnecessary to mention here all the various measures that should be adopted.

In the *direct treatment*, which must in most instances be conjoined with the causal, especially in the earlier periods, in consequence of the absence of any apparent *causa morbi*, electricity should always be, in the first instance, resorted to. The well-known and already frequently mentioned antispastic methods may here be successively tried. The anode may be kept steadily applied over the accessory nerve; muscular contractions may be repeatedly produced by frequent closures of the current; the current may be passed both longitudinally and transversely through the head and through the vertebral column in the cervical region, in which case the parts in the vicinity of the medulla oblongata, and those near the motor centres in the cerebral hemispheres, may be kept specially in mind; the galvanic current may be applied to the sympathetic in the neck, and to any pressure points that may be discovered; and lastly, gradually increasing faradic currents may be applied to the nerve and also to the muscles, and ultimately the skin may be irritated by the faradic brush;—all these may prove serviceable in particular cases, though only too frequently electro-therapeutic measures are ineffectual.

The subcutaneous injection of *narcotics*, and especially of morphia, atropine and woorara, are next worthy of trial, and in many cases serve to cure the disease. Narcotic salves and the application of wool, impregnated with chloroform or ether, may also be tried.

The true *nervines*, especially the valerianate of zinc, arsenic and bromide of potassium, may be used, and often prove serviceable, though too much should not be expected from them.

The application of *derivatives* in the form of sinapisms, flying or open blisters, moxæ and pustule-producing ointments, has recently received a new impulse from the favorable results which Busch has obtained in some chronic cases by the energetic application of the actual cautery. In several severe and obstinate cases of spasms of the muscles supplied by the accessorius, Busch applied a prismatic cautery to band-shaped portions of the skin, five or six inches in length, on both sides of the spinal column, in the cervical region ; after which he kept the patient quiet for some time, and, when the eschar separated, maintained suppuration for some weeks. In three cases permanent recovery took place, whilst in the fourth there was no improvement. These results, however, encourage us to make further trials in this direction.

Good results can only be expected to occur in slight cases from the application of cataplasms, from warm baths, and from gymnastic measures.

Operative measures have been adopted in desperate cases, and subcutaneous *myotomy* of the sterno-cleido-mastoid has been performed with good results in a number of cases.

Neurotomy of the accessory nerve is an objectionable proceeding, partly on account of the resulting paralysis, and partly because it has been tried and has failed in effecting a cure. Michel has performed it without permanent result, and Busch saw, after resection of a portion of the accessory, the spasms recur in the course of a few days with unabated violence ; the excitations proceeding from the centre were here evidently conducted to the muscles by the fibres running in the cervical nerves. Such an operation, therefore, could only be justified in cases where it is possible to demonstrate the existence of causes situated in the trunk of the accessorius itself.

2. *Tonic Spasm* (*and Contracture*) *of the Muscles Supplied by the Accessorius.*

This condition is, for the most part, limited to the sterno-cleido-mastoid, much more rarely to the trapezius ; occasionally both muscles are contracted.

If the sterno-cleido-mastoid be alone affected, the characteristic position of the head (the chin turned to the opposite side and raised ; the ear approximated to the clavicle) is maintained with greater or less persistence, and is difficult to rectify either by the patient's own efforts, or by passive movements. The muscle forms a strongly-marked, hard projection. This is the so-called *caput obstipum spasticum.*

Sharp pains for the most part accompany the earlier periods of the spasm ; these, however, gradually diminish, so that the permanent condition of contraction of the muscles is, as a rule, free from pain. The muscle itself occasionally becomes hypertrophied, but atrophy of the antagonistic muscles, which are no longer called into action, is more frequently observed. A permanent curvature of the cervical portion of the spinal column may be produced in young persons by the persistent oblique position of the head and neck, which becomes convex towards the sound side. Nothing is known of pressure points in this form of spasm.

In tonic spasm of both sterno-cleido-mastoids,—Duchenne has observed a case of this kind,—the head is drawn strongly forwards and is depressed upon the chest.

In tonic spasm of the trapezius the head is persistently inclined towards the affected side, the occiput is approximated to the shoulder, the shoulder itself is somewhat raised, and the scapula is drawn inwards. At the same time the anterior border of the trapezius forms a hard swelling. If the middle and lower fasciculi be chiefly affected (which is a rare occurrence), this is indicated by the corresponding position and rotation of the scapula. This bone is always fixed, under these circumstances, and its position cannot be changed by passive movements—a characteristic difference from the changes in position of the scapula caused by paralysis of the antagonistic muscles.

The *etiology* of this form of spasm is, if possible, still more obscure than that of clonic spasm. In many instances it either is congenital or it originates during infancy (probably from injury to the brain, "*Gichter*," etc.). It is sometimes a sequela of convulsive attacks. Severe forms of clonic spasm are occasionally observed to merge into tonic spasms, and thus to lead to a state of permanent contracture. Disease of the cervical vertebræ is not unfrequently accompanied by tonic spasm of the muscles supplied by the accessorius. The rapid transition into paralysis explains the importance of the early phenomena of irritation. The causes of this spasm are most frequently injurious influences of an atmospheric nature, as the various modes in which one may "catch cold" (torticollis rheumatica). Hence it may be a question whether the rheumatic affection is seated in the nerve or in the muscle, and it is probable that the two conditions may co-exist.

In the *diagnosis* of this affection it is only important to avoid mistaking it for paralysis of the antagonistic muscles ; this mistake, however, can readily be avoided by the exercise of a little care. The possibility of carrying out passive motions in paralysis is pathognomonic.

In the *treatment* it is necessary, above all things, to ascertain and remove the causes of the disease. Energetic treatment is of special importance in the early stages of the rheumatic forms, as by the establishment of free diaphoresis, by purging, by the employment of vapor baths, and by poultices. In regard to the direct treatment, *electricity* is here again indicated. Besides the methods mentioned above, faradisation of the antagonistic muscles has also been practised with success.

Little good can be expected in this form of spasm, especially when the contracture is persistent, from the employment of *narcotics* and *nervines.* It is useless therefore to spend much time upon them ; but resort may be had at a very early period to surgical and orthopædic treatment, which here gives exceedingly satisfactory results. In the mildest cases excellent results may be obtained from active and passive gymnastic exercises of the muscles, and from rectification of the position of the head by means of properly arranged stiff cravats lined with wool. In severe cases it is necessary to commence the orthopædic treatment by forcible extension of the contracted muscle, whilst the patient is under chloroform, or by the performance of subcutaneous myotomy. The combination of appropriate retentive apparatus, with faradisation of the antagonistic muscles and active and passive gymnastics, must aid in completing the cure. This can only be considered as perfect when the originally contracted muscles have recovered not only their normal length and extensibility, but also their normal reaction to voluntary impulses. It is only in extremely few instances, however, that this can be accomplished. Campbell saw good results follow section of the accessory nerve.

e. *Spasm of the Muscles supplied by the Cervical and Dorsal Nerves.* (*Plexus cervicalis and brachialis.*)

Very numerous and very various forms of spasm occur in this extensive region of nerve distribution. In most instances several and often widely-separated muscles, and not unfrequently large groups of muscles, obeying a common centre of innervation, are affected by the spasm. A strictly systematic account, indicating the special anatomically distinct nerves, would only lead to numerous superfluous, and, from the very nature of the case, unjustifiable subdivisions; we propose, therefore, to treat of these forms of cramps collectively, and the following natural groups need only be distinguished.

1. *Cramps of Individual Muscles and Groups of Muscles in the Neck and Back, in the Shoulder, and in the Arm.*

Romberg, l. c.—*A. Eulenburg*, l. c.—*C. Bell*, l. c. p. 345.—*M. Meyer*, Electricität. 3. Aufl. pp. 302, 312 et seq.—*Duchenne*, Electris. local. III. édit. 1872.—*Zuradelli*, su varie specie di contratture, etc. Gaz. med. ital. Lomb. 1861, Nos. 4–8.—*Remak*, über Krämpfe. Allg. med. Centralz. 1864. Nos. 23, 28 u. 29.—*Hitzig*, über reflexerregende Druckpunkte. Berl. klin. Woch. 1867, No. 7.—*Erb*, Krampf im Splenius. Arch. f. klin. Med. V., p. 520. 1869.—*Dahl.* Fall von begrenztem klon. Krampf. Hospit. Tid. 1872 (Virch.-Hirsch, Jahresber.)

Isolated muscular spasms, either tonic or clonic, are of rare occurrence in this region; they are recorded in literature only as curiosities; their etiology and pathology are equally obscure, and the treatment which they require must be conducted on general principles.

The most frequent and remarkable of these forms of spasm, those, namely, exhibited by the sterno-cleido-mastoid and trapezius, have already been separately described, and of the rest, the more important will now be mentioned, with a short description of their characteristic symptoms.

Spasm of the Splenius Capitis.—In this affection the head is drawn backwards and towards the affected side ; the chin is somewhat depressed, and directed towards the corresponding shoulder ; and at the spot where the splenius appears beneath the anterior border of the trapezius, a hard roll can be felt. (The diagnosis of this from spasm of the trapezius is founded on the fact, that in this last the head is rotated towards the opposite side. In spasm of the sterno-cleido-mastoid, the chin is raised and rotated towards the opposite side, whilst the mastoid process is drawn forwards and downwards.) This spasm is for the most part of a tonic character (with remissions and occasional spasmodic contractions, Erb), or appears in the form of a permanent contracture (Duchenne).

Spasm of the Obliquus Capitis Inferior.—Since the action of this muscle, when it is in a state of spasm, causes horizontal rotation of the head, the head is either rotated intermittingly (tic rotatoire) or (where the spasm is tonic in character) persistently around its vertical axis, without any elevation of the chin or depression of the mastoid process. Clonic spasms of this muscle are a very burdensome evil, the patient being obliged to fix the head, or to replace it in its natural position, with the hands, if he wishes to look at a fixed object, or to speak. He may also be observed to correct the oblique position of the head with the hand when walking.

Spasm of the Deep Muscles of the Neck is characterized by strong retraction of the head towards the back, when the affection is bilateral, or towards the affected side, when it is unilateral. It is of frequent occurrence, though the deep position of the muscles renders it generally impossible to indicate those that are specially implicated. The spasms are of both a tonic and clonic nature ; and the symptoms so frequently observed of "stiff-neck," and boring of the head into the pillow, and a large proportion of all the cases of cramps in the neck, are evidently due to spasm of the muscles at the back of the neck.

Spasm in the Rhomboidei is rendered evident by the peculiar position of the scapula, the lower angle of which is raised so that its inner border assumes an oblique position, running from within and below, upwards and outwards, whilst it is at the same time approximated to the vertebral column; the muscle can be seen and felt as a firm swelling between the spinal column and the scapula. The border of the scapula is not lifted away from the chest, which distinguishes this affection from paralysis of the serratus anticus; the shoulder is not depressed. A distinct resistance is experienced when an attempt is made to raise the arm to a vertical position, on account of the antagonism of the serratus anticus major. Up to the present time tonic spasm or contracture of this muscle has alone been observed.

Spasm of the Levator Anguli Scapulæ.—In this affection the scapula is strongly elevated, especially in its upper and inner angle, the head is slightly inclined to the same side, the shoulder is somewhat drawn forwards, the supraclavicular fossa is increased in depth, the muscle it contains projects distinctly beneath the anterior border of the trapezius, which can easily be isolated from it by faradisation. It frequently occurs in the form of a tonic contracture, in combination with spasm of the rhomboidei or of the trapezius.

Spasm of the Deltoid.—Here the arm is held out from the body, and in cases of partial spasm is directed sometimes forwards, sometimes backwards, the scapula being at the same time pressed backwards towards the vertebral column, as in paralysis of the serratus, from which, however, it can easily be distinguished. Such is the condition in tonic spasm and in contracture of the deltoid; in cases of clonic spasm the arm is thrown upward and moved convulsively in various directions; in most instances other muscles may be seen to be implicated.

Spasm of the Serratus Anticus Major, of the Latissimus Dorsi, of the Teretes, of the Supra- and Infraspinatus, of the Subscapularis, and of the Pectoralis Major, may as a rule be easily recognized by the characteristic positions and movements of the scapula and arm (elevation, depression, rotation, adduction, and abduction) in combination with distinct hardness and fulness in the regions of the several muscles affected. Careful and exact observation is almost always indispensable for determining the exact nature of the disease. Spasms of this kind, both tonic and clonic in character, and for the most part unilateral, though often also bilateral, are only rarely observed (see, for example, the remarkable case of bilateral clonic spasm of the pectoralis muscle recorded by Remak).

Spasm of the Muscles of the Arms occurs in very various modes and combinations. For the most part the muscles supplied by a single nerve trunk are affected with spasm, as occurs, for example, in the contractures which are not unfrequently met with in injuries to the nerves of the upper extremities, and which are usually accompanied by symptoms of paralysis and frequently also by neuralgia. They owe their origin partly to the inflammation established in the nerve itself, in consequence of the wound, and partly to secondary alterations of the muscular substance. In other cases the spasms affect various muscles irregularly, as in the temporary contractures described by Duchenne, which often occur in hysterical patients, in consequence of violent movements, or the contracture of the biceps and supinator longus described by Zuradelli, which occurs in consequence of rheumatism and articular inflammation. There are yet other cases in which more or less spasmodic irritation has been observed to affect the greater number of the muscles of the upper extremity, as in the cases of "ascending contracture" described by Duchenne, which often spreads over the greater part of the upper extremities, in consequence of irritations in the joints ; as in the cases of tremor limited to the arm, of which M. Meyer has described two ; as in the cases mentioned by Busch of clonic spasms of one arm consequent on neuralgia ; and as in the cases described by Dahl of violent clonic spasms limited to the forearm, which were probably due to central disease. I have also myself observed a case of unceasing clonic spasms in one arm in which, after it had lasted for a long period, spasm of the tongue and face supervened, and which probably was of centric origin. Weir Mitchell has described spasmodic movements in stumps left after amputation of the arm ; they occasioned unceasing movement of the stump, and were regarded by him as of reflex origin. To this category belong also the cases in which various general forms of spasm are localized by preference in one or both upper extremities; as, for example, in tremor alcoholicus and paralysis agitans, or where epileptic attacks are preceded by a convulsive aura in one of the upper extremities; lastly, the spasms which not unfrequently appear in the upper limbs as subsidiary symptoms of central paralyses and lesions, which are only here referred to for the sake of completing the subject.

The symptoms of spasm that may present themselves in the muscular regions to which we have referred are innumerable, as are also the combinations in particular cases of the muscular regions affected with spasm, so that it is rare to meet with two cases that are in all respects alike. Two short examples may illustrate these remarks:

A woman, aged 42, dates her disease from a journey, taken shortly after a miscarriage, and in the course of which she took cold. For about nine months she has suffered from spasms in the neck, which have gradually increased in intensity, and led to her present lamentable condition; the head is constantly kept strongly inclined backwards by the spasm, and tossed to and fro, so that vision is seriously interfered with. The *muscles of the neck and small occipital muscles* are chiefly implicated, whilst the head is inclined backwards with great force, and rotated hither and thither; the strong contraction of the muscles can be distinctly felt beneath the occipital bone. The head is turned somewhat to the left, and if an attempt is made to restore it to its natural position, remarkable resistance is experienced, which gradually rises in intensity, and is followed by an increase of the spasm. The same is observed when the patient makes a voluntary attempt to keep the head still. In addition to the cervical muscles, the trapezii present partial contractions, as do also the right sterno-cleido-mastoid, and various muscles of the face. The arms are unaffected; acute pain is felt in the neck and shoulders. There is a sensation of constriction in the throat. The patient has a great inclination to yawn; and if she accomplishes this, which she is for the most part unable to do, the spasm is relieved for a little while; the spasm ceases during sleep, but difficulty is experienced in getting to sleep. Fatigue, emotions, and cold weather make it worse. No pressure points can be discovered. In this case considerable improvement resulted from the employment of galvanism, and the administration of the valerianate of zinc with iron.

Catharine Weiss, æt. 22, had convulsions at the age of four years; from her thirteenth year she held her head obliquely, especially while working; in her fifteenth year she had an acute affection of her central nervous system (inflammation of the brain), which confined her to her bed for three months, since which time her present malady has constantly increased. The extremities of the left side are somewhat weaker than those of the right. She suffers from headache; her menstruation is regular, but often painful; the position maintained by the patient is very remarkable: the head is held obliquely, inclined forwards, the chin depressed and strongly rotated to the left, just as if she wished to touch the shoulder with it; the shoulder is raised and drawn somewhat forward; the arm is rotated inward and strongly adducted. Looked at from behind, the upper part of the back appears to be slightly rotated around a vertical axis to the right, the head so strongly rotated to the left that the left ear is directed backwards, and the whole left side of the face is visible from behind; the patient is able to bring the head into a tolerably straight position by a voluntary effort, but it very soon returns to its abnormal position by means of a series of spasmodic movements; and the same is the case with the arm, the elbow-joint of which is usually maintained in a bent position. Manual labor decidedly increases the spasm. Careful investigation shows that the following muscles undoubtedly take part in the spasm:—the left splenius, the levator anguli scapulæ, the anterior part of the left trapezius, and in addition the scaleni, the obliquus capitis inferior, the pectoralis major, the teres major, and the biceps brachii of the left side; in all probability there are yet other muscles, especially the deep cervical muscles lying on the anterior surface of the vertebral column, inaccessible to palpation and precise investigation, which participate in the spasm. The cutaneous sensibility is quite normal. No pressure points are discoverable. The motility of the left upper extremity is not materially impaired; the lower extremities are normal. The cerebral nerves are also otherwise normal. The patient was treated electro-therapeutically in every possible way, but received no permanent benefit.

In regard to other symptoms the reader is referred to the description of spasm of the muscles supplied by the accessorius, to which the forms of spasm here under discussion are in every point of view closely allied. The feeling of exhaustion, the difficulty of getting to sleep, the cessation of the spasm during sleep, its increased intensity during standing, walking, and other occupations, are all characteristic features of both affections. In a few instances pressure points, both exciting and inhibiting, have been discovered, and favorable results have been obtained by the application of remedial agents to them.

In regard to the *etiology* of the affection, we may also refer to what has been already said in the chapter on spasm of the muscles supplied by the accessorius; here also the causes may be either central or peripheric, and it may be due to rheumatism or to some kind of reflex action; in many instances no obvious cause can be discovered, so that we are often obliged to refer spasm, limited to particular muscles, to a nervous diathesis, to hysteria, or to some similar and not very intelligible cause.

The *diagnosis* may present difficulties of various kinds. In the first place, it is not easy to determine the particular muscles affected, and, as it is impossible here to enter into details, we can only recommend that the examination of the patient should be made as carefully as possible with the assistance of active and passive movements, local faradisation, etc. The chief points on which the diagnosis of the more important forms of spasm must rest, have already been given. The distinction between spasm of a muscle and paralysis of its antagonists does not in general present any great difficulty, since positive criteria are supplied by the possibility of passively moving the parts, and by the effects of local faradisation. The determination of the seat and point of origin of the disease is in many cases remarkably difficult and sometimes impossible, and our conclusions must be based upon the general principles of diagnosis and upon the results of a thorough and complete examination of the patient. Our extreme ignorance of the essential nature of convulsions, however, does not enable us to proceed very deeply into the subject, but it may be stated that in the greater number of cases the disease appears to be due to centric lesion.

The *prognosis* must be founded on general principles, and must depend upon the seat, causes, and duration of the disease, and upon the character and constitution of the patient; no further observations need be made on this point.

Were we to enter into any details in regard to the *treatment* of these forms of spasm it would only lead to unprofitable repetition, and it will be sufficient to refer to what has been said in describing the treatment of spasm of the muscles supplied by the accessorius. The effects of the remedial means there mentioned are equally doubtful in both cases; it may, however, be mentioned that Duchenne has obtained particularly successful results, in cases of contracture of the several muscles, by faradisation of their antagonists, and that he strongly recommends cutaneous faradisation in cases of recent rheumatic contractures. In a case of violent clonic spasm of one arm, Busch succeeded in removing the spasm by means of the application of the actual cautery to the back, though the primary neuralgia was not thereby improved.

2. *Spasms of the Respiratory Muscles.—Inspiratory and Expiratory Cramps, Singultus, etc.*

Romberg, A. Eulenburg, M. Rosenthal, l. c.—*Benedict,* l. c.—*C. Bell,* l. c.—*Duchenne,* Electris. local. II. u. III. édit.—*M. Meyer,* Electricität, 3 Aufl.—*B. Brodie,* Lectures illustrative of certain local nervous affections, London, 1837.—*R. Bright,* Spasmodic diseases accompanying affections of the pericard. Med. chir. Transactions, XXII. 1839.—*G. Hirsch,* Spinalneurosen. 1843, pp. 230, 250. —*Oppolzer,* tonischer Krampf des Zwerchfells. Spitalszeit. 1862, No. 24.—*Biermer,* über Bronchialasthma. Volkmann's Sammlung klin. Vortr. No. 12, 1870.—*Klein,* Singultus vicarirend f. Husten. Deutsch. Klin. 1857, No. 37. —*Lanquaille de Lachèse,* Hoquet intermittent. Gaz. hébdom. 1865, No. 4.—*Dumontpallier,* Cas rare de hoquet nerveux. Union méd. 1867, No. 150.—*Carcassonne.* Hoquet très grave. Paris, 1868.—*Ferber,* d. Niesekrampf, etc. Arch. d. Heilk. X. 1870.—*Mosler,* Fall von Niesekrampf. Virch. Arch. XIV., p. 557, 1858.

The respiratory muscles are not unfrequently affected spasmodically, and the fact that many muscles lying widely apart and innervated by very different peripheric nerves are coincidently affected by spasm renders it probable that such spasms are of centric origin. Nevertheless, the diaphragm, which is the chief muscle of inspiration, may also be alone affected, and this is undoubtedly the commonest form of respiratory spasm. It therefore appears advantageous on practical grounds to consider the several special forms separately.

Tonic Spasm of the Diaphragm.—This affection is, upon the whole, rare, but is accompanied by very alarming symptoms. Duchenne first pointed out the symptoms to be expected in this form of spasm from his physiological experiments; and the cases afterwards observed by himself, Valette, Vigla, Oppolzer, and others have corroborated his statements in all essential particulars. Patients affected with this form of spasm exhibit the most intense dyspnœa, and are threatened with asphyxia. The lower half of the chest is strongly expanded and immovable, the epigastrium projects strongly, whilst rapid and superficial respirations are performed with the upper part of the chest. The intense dyspnœa compels the patient to sit up in bed ; well-marked cyanosis appears, the voice becomes feeble and muffled, and severe pain experienced in the epigastrium and along the attachments of the diaphragm increases his misery. If the attacks last for a long time death may ensue, and even in moderately violent cases the symptoms of asphyxia make their appearance in the course of a few minutes.

The attacks present a certain similarity to bronchial asthma, and some observers have been induced, by the demonstrably low position which the diaphragm maintains in this last affection, to assume a tonic spasm of this muscle as the cause of bronchial asthma (Wintrich, Bamberger). There are, however, characteristic differences between the two conditions, and the frequent respiration (with *short* expiration), and rapidly supervening asphyxia in tonic spasm of the diaphragm, as well as the movements of the muscle, which, according to Biermer, although limited, are always present in bronchial asthma, will usually enable us to make the diagnosis.

This form of spasm has been hitherto observed to occur either in consequence of catching cold, or as a complication of muscular and articular rheumatism, and on this account it has usually been regarded as a rheumatic affection of the diaphragm itself. To what extent this is correct cannot at present be determined, on account of the few cases that have been observed. It constitutes, in some instances, a complication of tetany, and in tetanus it is not unfrequently the immediate cause of death by asphyxia.

The *treatment* must be extremely energetic, as a few minutes may determine the issue of life or death. Amongst the remedies to which it is best to resort are inhalations of chloroform, subcutaneous injections of morphia, and strong irritation of the skin, such as the application of compresses dipped in hot water, the application of the faradic brush (with a strong current) in the neighborhood of the diaphragm, and the application of galvanic and faradic electricity to the phrenics.

Clonic Spasm of the Diaphragm. Singultus. Hiccough. (German, *Schluchzen ;* French, *Hoquet.*)

Every one is familiar with the peculiar and often very dis-

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tressing contraction of the diaphragm, termed "hiccough," which is repeated for a certain length of time, with intervals of various lengths, and which, when of moderate intensity and duration, may fairly be regarded as physiological. It may, however, rise to such a pitch of intensity, and last so long as to deserve to be classed as pathological, and to become a serious annoyance to the patient. In such cases energetic, short, spasmodic contractions of the diaphragm occur, accompanied by an inspiratory sound, which is usually suddenly arrested by the closure of the glottis. The several contractions may succeed one another more or less rapidly, often indeed so rapidly that more than a hundred contractions occur in a minute. The attacks may last for hours, days, or weeks, and may recur more or less frequently for years. When violent, they cause severe pain in the epigastrium and along the attachments of the diaphragm, and the epigastrium is drawn in with each contraction. The rhythm of the respiration is considerably disturbed, and dyspnœa occurs if the hiccough be very frequent. Speech is disturbed, and there are usually nervous conditions affecting the system at large. The spinous processes of the cervical vertebræ are generally sensitive to pressure. Great difficulty is experienced in getting to sleep, and the spasm is not always arrested during sleep. The ingestion of food is interfered with, digestion is imperfect, and the malady has some influence even upon the circulation. Danet observed hiccough to occur synchronously with a very slow pulse.

The *etiology* of hiccough is very manifold; in some rare instances it seems to be caused by direct irritation of the phrenic nerve, but, in all probability, the spasms would partake rather of a tonic character in such a case. On the other hand, direct irritation of the respiratory centre, or of the fibres of the phrenic in their course through the spinal cord, is much more likely to be the cause. Hiccough has been observed in diseases of the central nervous system, and in injuries of the skull and cervical portion of the spinal column. The influence of anæmia and chlorosis, of cachexia, of hysteria (which, according to Benedict, is very common in Jewesses), of malarial poison, of emotions, such as fear and grief, in producing hiccough, may be explained on the same principle. Hiccough is, however, far more frequently caused by reflex action proceeding from the subjacent viscera; simple repletion of the stomach, or pressure upon this organ, is often sufficient to cause it, and it is often seen in cases of gastric and intestinal diseases of all kinds: in peritonitis, in hepatic and uterine affections, in disturbance of menstruation, and in affections of the prostate gland. The untimely arrest of diarrhœa, the action of an emetic, may produce it; more rarely it accompanies other diseases, as pericarditis. Klein saw it follow a prolonged fit of coughing. It is oftentimes an ominous sign in severe cachectic conditions, especially in cancer of the abdominal viscera.

In all these cases we are almost entirely ignorant of the real cause and nature of this peculiar, periodically recurring spasm, which is in fact a true spasm of the diaphragm.

The *prognosis* is in most cases favorable (though of course without reference to the prognosis of the primary disease). In most instances this troublesome symptom may be removed, though it often resists all treatment in the most obstinate manner, as is especially observed in well-marked hysteria and similar serious nervous affections; the final singultus in cachectic conditions is also difficult to arrest or cure.

The *treatment* of hiccough often presents considerable difficulty, requiring the adoption at times of extraordinary means. As a general rule, the causal indications must first be met; but when this is impossible, or when treatment directed in accordance with these indications proves ineffectual, resort must be had to direct treatment, and innumerable suggestions have been made with this object in view.

In slight cases *psychical impressions*, such as sudden fright, diversion of the attention, excitement, are sufficient ot stop the affection. Probably Cruveilhier's successful plan of pouring water into the mouth till the patient feared he was about to be suffocated, belongs to this class. It should be remembered, however, that this procedure must have produced irritation of the superior laryngeal, which leads by reflex action to relaxation of the diaphragm. To the same influence we may attribute the success of all those methods which occasion powerful movements of expiration, such as sneezing, long retention of the breath, and straining with closed glottis.

In severe cases, violent peripheral irritation may be practised, and this may be accomplished in various ways, as, for instance, by the inhalation of strong odors, by burning blotting-paper or feathers under the nose, by sinapisms and the application of scalding hot fomentations or vesicants over the diaphragmatic region. Of all these means the faradic brush is unquestionably the most effective, when applied of full strength to the epigastrium and hypochondria. I have myself obtained excellent results with it in the case of an old cachectic man. Strong irritation of the region supplied by the superior laryngeal nerve is particularly worthy of trial. *Electricity* is also very effective when applied directly to the phrenics and to the neighborhood of the respiratory centres. Eulenburg was very successful in curing a severe case by galvanization, and Dumontpallier in another case by faradisation of the phrenic nerves. Galvanization of the neck and the transmission of a constant current through the mastoid processes has also proved serviceable. The *narcotics* appear to be of great value in this affection, and amongst them opium, cannabis indica, and especially the subcutaneous injection of morphia, are worthy of trial. Benedict strongly recommends the exhibition of atropine. The inhalation of ether and chloroform, as well as the internal use of these anæsthetics, has also been highly praised.

In desperate cases the action of the various nervines and antispasmodics should be thoroughly tested, since they often prove extremely serviceable. Amongst these, zinc, valerian, assafœtida, arsenic, strychnia, and nitrate of silver may be mentioned. Klein saw a case in which a cure was effected by means of musk in combination with alkaline baths. Lastly, it may be mentioned that Carcassonne saw good results follow the introduction of a sound into the œsophagus in a very obstinate case, and that circular compression of the base of the thorax, with forcible flexion of the head upon the chest (for a period of from five to ten minutes), has been recommended as a cure for hiccough.

Inspiratory Spasm. Spasmus Inspiratorius.—By this term is understood (in opposition to the above described isolated contraction of the diaphragm) a spasmodic rhythmic action of all or almost all the inspiratory muscles—a spasmodically increased inspiratory effort, both as regards depth and frequency. The diaphragm is of course implicated, and in fact all degrees of the affection are observed, from simple hiccough to complete spasm of the inspiratory muscles ; but cases also occur in which hiccough as a symptom is either not well marked or is entirely absent; and, moreover, true inspiratory spasm differs very distinctly from simple hiccough. The essential feature of the affection is that either many or all of the muscles of inspiration participate in the spasm, and that a true inspiration, unbroken by sudden closure of the glottis, takes place. Obviously, therefore, the dilatation of the glottis, which belongs to a true inspiration, also occurs.

The spasm consists in a more or less rapid and long sequence of deep inspirations, whilst the intervening expirations are performed in the usual easy and noiseless manner. The chest is powerfully expanded by the energetic contraction of the inspiratory muscles, the epigastrium is protruded, or, if the spasm be very intense, is pressed inwards by the pressure of the external atmosphere, the auxiliary muscles of respiration are excited to action ; at each inspiration the pectorales and sterno-cleido-mastoids are brought into strong relief, the shoulders are raised, the head is drawn backwards, and the respiratory muscles of the face, alæ nasi, and eyelids contract. In many instances, a loud hooping sound, which rarely resembles that observed in hiccough, accompanies inspiration, and this is often followed by the peculiar gurgling sound, which ordinarily accompanies the ascent of air in the œsophagus (eructation). The origin of this sound is readily explained by the fact that the abdominal viscera are powerfully compressed by the energetic inspiratory movements ; in consequence of this a portion of the air contained in the stomach, which is full of it, is pressed through the cardia into the œsophagus and rises with a gurgling sound. In those who are much troubled with eructation it may not unfrequently be observed that each act is preceded by a short spasmodic inspiration.

The efficacy of the respiration is more or less disturbed by the violence of the spasm, and swallowing is rendered difficult ;

dull pain is experienced in the epigastrium, the abdomen is usually tympanitic, and there are as a rule other symptoms of nervous derangement, especially of hysteria. The spasm usually occurs in paroxysms of variable duration, but may continue without interruption for weeks or months. Relief always occurs during sleep.

A spasm of this nature occurred in a robust tradesman, aged 45. It had lasted for two years, and no cause could be assigned for its appearance. The attacks came on more or less frequently, and especially after emotional disturbances and unusual exertion. Each attack consisted of a spasmodic contraction of a large group of the muscles of inspiration, lasting for one or two seconds, and accompanied by a hooping noise. The shoulders were strongly raised, the head was drawn backwards, the chest was expanded, and the epigastrium was depressed during the stronger spasms. The participation of the trapezii, the sterno-cleido-mastoids, the scaleni and both platysmata in the spasm was very evident. The attacks were usually preceded by a very peculiar and unpleasant sensation in the cardiac region, a sensation which was particularly well marked when the stomach was empty.

A healthy looking girl, 19 years of age, affected with migraine, but without hysterical symptoms, had been suddenly attacked three weeks previously by vomiting, which was followed by the *inspiratory spasm* from which she was then still suffering. The patient was under the care of Friedreich, and exhibited a peculiarly rapid inspiration of a spasmodic character, in which the accessory muscles of inspiration participated, which lasted through the greater part of the day, but ceased at night. The epigastrium was strongly thrown forwards on each occasion; a hooping noise like that of hiccough accompanied each inspiration, and was regularly followed by the accompanying gurgling sound, characterizing the regurgitation of air in the œsophagus; no pressure points could be discovered. The spasm disappeared in a short time without the employment of any energetic treatment.

An hysterical patient, thirteen years of age, who had already repeatedly suffered from persistent aphonia and numerous other hysterical symptoms, presented the characters of a very severe inspiratory spasm, which had lasted for fifteen weeks uninterruptedly, excepting at night, when it ceased. Rapid and deep inspirations, accompanied by a loud snoring sound, followed one another uninterruptedly. The scaleni, sterno-cleido-mastoids, and trapezii, as well as the pectorales, were rendered very tense, and the muscles of the alæ nasi and of the eyelids contracted at the same time. The upper part of the chest was expanded, whilst the lower part, with the epigastrium, was drawn in. After some of the inspirations air escaped with a gurgling sound through the œsophagus. When the inspiratory spasm coincided with the systole of the heart, the pulse in the radialis became imperceptible. The belly was very much swollen, great difficulty was experienced in swallowing; there was complete aphonia, anæmia, &c. The further details of this case, which occurred in Friedreich's clinic, may be omitted here.

Biermer describes (loc. cit., p. 50) a case in which peculiar asthmatic attacks supervened, after a succession of spasmodic tetanic inspirations, which lasted from a quarter of an hour to an hour. Each inspiration lasted from two to four or more seconds, and was then succeeded by a rapid and loud expiration; acute pain was felt in the region of the diaphragm. There was secondary emphysema and bronchiectasis.

A peculiar form of *respiratory spasm* may frequently be observed in women who are either predisposed to, or who actually suffer from well-marked hysteria. It is characterized by an uncommonly rapid and spasmodic series of short, deep, hooping and sighing respirations (reminding the hearer of the panting of a hot and hunted dog), in which the epigastrium and the whole abdomen are thrown into strong to-and-fro movements. Both the expiration and the inspiration are spasmodically increased in intensity. The whole attack lasts for a minute or two, and recurs more or less quickly; it is not unfrequently associated with fits of laughter or crying, or with other hysterical symptoms.

It occurs especially from irritation of the generative organs, or as a result of uterine disease, or of unsatisfied sexual excitement, and during menstruation. I have seen a somewhat more persistent case of this form of spasm in an hysterical girl of eleven years of age, whose intellect was also somewhat disturbed.

The following are more complicated forms of respiratory spasm:

Attacks of Sneezing. Sternutatio convulsiva. Ptarmus.—These occur in a paroxysmal and spasmodic form. The patient will sometimes sneeze several hundred times in succession. (Mosler, in one instance, calculated that the patient had sneezed about 48,000 times in 80 hours!) The phenomena of sneezing are well known. There is first a deep inspiration, which is accompanied by a peculiar sensation in the nose, and is followed by a violent explosive expiration, the air being chiefly expelled through the mouth. It is a reflex act, usually produced by a stimulus acting upon the nasal mucous membrane. Attacks of sneezing, when of long duration, are productive of great misery to the patient, interfering with respiration, the ingestion of food, and digestion, and causing violent pain in those muscles that are chiefly brought into action, and dangerous exhaustion. The attacks are usually accompanied by a profuse watery secretion from the nasal cavities.

Attacks of *yawning* (*Oscedo, Chasmus*) consist of a series of yawns, which in pathological cases are extremely distressing and exhausting. Each consists of a deep, slow, noisy inspiration performed with widely opened mouth, and peculiar contraction of the muscles of the buccal cavity and throat, which is followed by a similar loud, noisy and protracted expiration. The contagious effect of the idea or the sight of others yawning is well known.

Under the term *spasmodic cough* are comprehended all those paroxysmal attacks of coughing, which are accompanied by a loud ringing sound. The cough is a sudden spasmodic expiration performed with relaxed diaphragm and contracted glottis, and generally results from irritation of the sensory fibres of the superior laryngeal nerve. Such attacks of coughing may last for a variable period, and may also recur frequently for months or years. The muscles of expiration may be called into play to a very extraordinary extent, without any apparent exhaustion being produced.

Fits of laughing and *crying* are both essentially forms of expiratory spasm. In the former there are sudden loud expirations, accompanied by vocal tones, with or without an expression of hilarity in the face; in the latter there are long-drawn expirations, often interrupted by sobs, accompanied by wailing or moaning sounds, and usually by a profuse secretion of tears. These peculiar forms of respiration are ordinarily the expression of very definite psychical states, but in pathological conditions they may often be quite independent of such states, and even arise in sharp contrast to them, and are then designated as spasmodic. These spasms usually occur in attacks of longer or shorter duration, which recur with variable frequency.

Our knowledge of the *pathogenesis and etiology* of the above-named forms of spasm is still very imperfect. Physiology has not yet explained the finer mechanism of the more complicated movements of respiration sufficiently to afford an insight into their pathological conditions, and even well-ascertained physiological facts are probably not precisely applicable to the explanation of simple respiratory spasms. At any rate, the physiological stimuli for the inspiratory centres (deficiency of oxygen and the accumulation of carbonic acid) are probably, under pathological conditions, *not* the excitors of spasmodic respiratory movements. In all these forms of spasm, however, the various respiratory centres are excited either directly or by reflex action. We have no more exact knowledge upon this point.

Moreover, these kinds of spasm scarcely ever occur in an isolated form, but in the greater number of cases are complicated with other nervous disturbances, or constitute subordinate symptoms of severe general neuroses, and especially of hysteria, or they constitute a symptom of disease of the central nervous system; thus laughter, crying, and spasmodic yawning are not unfrequently accompaniments of cerebral diseases, apoplexy, etc. Hysteria especially presents examples of all these forms of spasm, which in that disease often succeed one another with extraordinary variety. Reflex irritation is often a cause; thus it may be produced by uterine diseases, pregnancy, worms, skin diseases, sexual excitement, disturbances of the circulation in the true pelvis, hemorrhoids, dysmenorrhœa, etc.; and these peripheric causes are particularly frequent in attacks of sneezing, and are seen in irritation of the nasal mucous membrane (coryza, hay asthma, etc.), and the conjunctiva, in disease of the ear (Mosler), in sexual excitement, and in hemorrhoids. A definite connection can be traced between this form of cramp and migraine, whooping-cough, and bronchial asthma (Ferber).

In the *treatment* of these forms of spasm, attention should first be directed to the removal of the cause, and this most frequently is hysteria. (See the section devoted to this subject.)

In the direct treatment of inspiratory spasms, the same measures must be adopted as in the treatment of hiccough. In the more complex forms a selection must be made, according to the circumstances of the case, between cutaneous irritants, electro-therapeutic measures, narcotics, and anti-spasmodics. For the cure of sneezing, which, it would appear, most frequently requires energetic measures, compression of the root of the nose (?), plunging the head into cold water, strong irritation of the skin, emetics, inhalation of chloroform and of the vapor of iodine through the nose, may be successively tried, together with the local application of solution of quinine (in hay asthma), and of narcotic solutions or snuffs, etc. Mosler effected a cure in one case by resorting to strong diaphoresis (by means of hot baths), cutaneous irritants, and opiates.

3. *Writers' Cramp (Schreibekrampf).—Graphospasmus.—Mogigraphia. (Pianists' Cramp; Tailors' Cramp; Milkers' Cramp, etc.)*

Text-books of *Romberg*, *Hasse*, *A. Eulenburg*, *M. Rosenthal*, *Benedict;* Manuals of Electrotherapeutics.—*Brück*, Hufeland's Jour. 1835. St. 4.—*Stromeyer*, bayr. med. Correspondenzbl. 1840, No. 8.—*G. Hirsch*, Spinalneurosen, 1843.—*Fritz*, über Reflexionsfingerkrampf. Oesterr. Jahrb. 1844. Bd. 46 u. 47.—*Cazenave*, de quelques infirmit. de la main droite, etc. Casp. Wochenschr. 1848, No. 16.—*Thielmann*, Fall von Nähekrampf., Med. Zeit. Russlands, 1859, No. 44.—*Haupt*, über d. Schreibekr. Wiesb. 1860.—*Tuppert*, z. Behandl. des Schreibekr., Bayr. ärztl. Intelligenzblatt, 1860, No. 24.—*Cris. Zuradelli*, del crampo degli Scrittori., Gaz. med. ital. Lomb. 1857. Nos. 36-42.—Annal. univers. 1864.—*Remak*, klin. Mittheil.—Oesterreich. Zeitschrift für prakt. Heilk. 1860, No. 45.—*Duchenne*, Spasme fonction. et. paralys. musc. fonct. Bull. de thérap. 1860. Electris. localis. III. ed. 1872.—*Solly*, Lectures on Scriveners' palsy. Lancet, 1865, Jan., and 1867, May.—*M. Meyer*, z. Ther. des Schreibekr.—Vers. d. Berl. ärztl. Ges. I. 1867.—*Runge*, z. Genese. u. Beh. des Schreibekr. Berl. klin. Woch. 1873, No. 21.

Under the term "Writers' Cramp," a large number of essentially different affections have been included, which have only this in common, that in writing and in other complicated and delicate occupations, such as sewing, piano-forte playing, drawing, etc., a disturbance of the requisite movements is apt to occur, whilst the coarse muscular movements are as a rule performed quite normally.

In writing, in particular, a large number of muscles are kept in constant, strained, and unceasingly modified activity; it is especially the small muscles of the hand (the interossei and lumbricales, the muscles of the thenar eminence, and the long flexors and extensors of the fingers) which are thus brought into action. It is unnecessary here to enter into minute details in regard to the small movements that produce the several strokes, which, when combined, form writing, since Zuradelli has discussed this subject very fully in his first work, and still less since in different individuals marked differences are observed, some writing more with the joints of the fingers, some more with the wrist, or even with the lower arm. The characters of writing, like the notes in playing the piano, or the movements required for sewing, are formed essentially by the association, consequent upon long practice, of a large number of very fine degrees of contraction of the small muscles of the hand, which are almost involuntary, and which follow one another with extraordinary rapidity; there is in addition a definite movement of the whole hand to the right, partly due to a movement of the upper arm (with rotation outwards, and a backward movement), and partly to the gradual extension of the forearm. The resultant of these two motions is the line on which we write.

Every one is aware of the trouble and pains that are required to learn the art of writing, and how much practice it takes to acquire a quick and fluent hand. Ultimately, however, the association and co-ordination of the several movements become so firm and certain that they follow one another almost or quite involuntarily.

It is probable that the co-ordination of these voluntary impulses occurs at different and definite points of the central organs (apparatuses of co-ordination), or it may be that the associated excitations of the will pass at certain points of the gray substance over routes which, from long and frequently repeated exercise, offer only a small resistance; so that ultimately the excitations of the will pass at once along these co-ordinating fibres. It is only in a normal condition of these fibres (conductivity and excitability normal) that natural writing (or similar complicated and well-practised movements) is practicable, and these fibres appear to a certain degree to be independent of the motor paths which serve for the conduction of the simple uncomplicated excitations of the will. It is not easy on any other view to understand how simple muscular actions can still be performed with facility, whilst the same muscles refuse to play their part in complicated associated movements.

It is clear that an harmonious co-operation of all the individual movements is absolutely requisite in order that the act of writing (or analogous acts) should be performed with the usual facility, and it is equally evident that disturbances of this harmony, which is the result of long practice, may proceed from very different points of the apparatus in question.

The points of greatest importance are perhaps the condition and behavior of the co-ordinating apparatus, since every change in its capacity for work and excitability will exert an influence in disturbing the co-ordination of movements ; every pathological increase or diminution of the resistance in particular paths of conduction will exhibit itself in some alteration in the muscular contractions at the periphery. If the excitability and functional activity of particular fibres be augmented, convulsions will be produced in the muscles, even when the voluntary impulses have undergone no change in strength. On the contrary, if the resistance in certain paths be increased, feeble action of the muscles will be observed, and a compensating increase in the energy of the impulses of the will will occasion convulsion or spasm in the associated muscles. If the co-ordinating apparatus be easily exhausted, arrest of the associated movements will occur after a short period of activity, whilst the ability to perform simple uncomplicated movements may still be preserved. As all these matters play an important rôle in many of the forms of writers' spasm (and similar forms of disease), Benedict has proposed for these neuroses the term of "co-ordinated business neuroses, or neuroses from occupation" (co-ordinatorische Beschäftigungsneurosen). The co-ordination of motion is, in point of fact, disturbed in most of the forms of disease thus produced, though certainly not always from disease of the co-ordinating apparatus (or of certain parts of the central gray substance), and it would only be by an unjustifiable extension of the idea of disturbances of co-ordination were all the disturbances of co-ordinated movements, caused by spasm or paralysis of particular muscles, to be designated by this name.

Peripheral disturbances may have a similar influence on the power of writing and upon the performance of analogous acts, for if it happen that particular peripheral nerves and muscles are more excitable than natural they will pass into a state of spasm, whilst if they are less excitable or paralyzed, the absence or defective degree of their contraction will likewise lead to a disturbance of the co-ordinated movements, provided, always (as indeed is not otherwise possible), the strength of the motor excitation liberated for the whole group of muscles remains the same.

In addition, it may be remarked that the sensibility of the hand, the so-called sense of muscular effort, and muscular sensibility probably play a not unimportant part in writing, especially in guiding and holding the pen. Disturbances in these organs, such as hyperæsthesia and anæsthesia in the centripetally conducting paths, may likewise lead either by direct or reflex action to disorder of the complicated mechanism required for these movements.

It is obvious that under the head of "writers' spasm" are included very various morbid conditions, having this only in common, that they disturb or render impossible the delicate and complicated movements required for writing. We are unable, at present, to classify, with any degree of accuracy, the several forms in accordance with their genesis, whilst the symptom of disturbance of, or interference with, particular avocations of life, which is common to them all, keeps them sufficiently together in practice. It is always advisable, however, with a view to obtain better and more exact knowledge, to distinguish the several forms from each other, and the three divisions suggested by Benedict are well adapted for this purpose, namely, the spastic, the tremulous, and the paralytic form.

Symptoms.—The disturbances of movement which render writing, or similar acts, difficult or impracticable, are highly characteristic. Such disturbances are in the first instance only slight, and are only perceived when the effort has been long-continued, and is then felt as a sensation of extreme weariness. By degrees the symptoms become more and more marked, and make their appearance very soon after the commencement of the exertion, and ultimately, as soon as the pen is taken in hand, or even when the hand is merely placed in the required position. The disturbance is increased if the attention be directed to it.

Close observation shows that considerable variations occur in the phenomena in different instances. True *spasmodic disturbances* (*spastic form*), in which there are tonic or clonic spasms of one or several muscles, are the most typical and, perhaps, the most frequent. In the earlier periods of the affection only slight spasmodic movements of particular fingers, with here and there an irregular stroke in the writing, are to be observed. After a time the spasms become stronger, and are generally tonic in character, affecting, usually, the thumb and first finger; they appear in the form of a sudden extension of the fingers, causing the pen to be dropped; or there is a spasmodic action of the opponens pollicis, with abduction and coincident flexion of the index finger, so that the pen is rapidly moved away from the paper; or a spasmodic flexion of the first three fingers occurs, so that they are pressed tightly against the pen, which cannot then be moved further onwards; or, there may be movements of pronation or supination in the forearm so that the pen is raised from the paper and moved backwards and forwards in the most irregular manner; or lastly, the abnormal movements of the pen may be occasioned by spasms of the muscles of the shoulders. More rarely, when any attempt to write is made, well-marked trembling of the hand and forearm is experienced, and at a later period, even of the whole arm, so that the pen only makes undulating or angular strokes, and the writing becomes completely illegible (*tremulous form of spasm*).

In all these cases the act of writing is rendered materially slower and more difficult, the formation of the strokes is retarded, as if the movements of the hand were forcibly resisted; and, as the spasm increases, the pen at length refuses to move—a pause must be made—after which the whole scene is repeated. The position which the patient often assumes, in order to facilitate his writing, and the means he employs to prevent the occurrence of the spasm, are often extraordinary. One will only rest the wrist on the paper, raising the elbow in the air; another supports the arm on the elbow, and writes with the wrist raised and free; another steadies the right hand with the left; another takes the pen between the index and middle finger, or sticks it into a cork, which he seizes with the whole hand. It is instructive to notice the changes that occur in the handwriting, consequent on disease, and a comparison may be instituted with letters formerly written in health; it is often quite altered in character, because the patient has adopted a new method of using the pen, when writing is possible. The strokes are coarse, imperfect, and unequal, and numerous irregularities and false strokes are to be observed; in the highest degrees of the affection, after a few scarcely legible words, the whole writing becomes a mass of irregular strokes and curves, whilst in other instances the letters are mere trembling, undulating or zig-zag strokes.

In strong contrast with the cases just described, are those in which fatigue and weakness of the hand and forearm constitute prominent symptoms, in which there are no distinct spasms, but in which paralysis is more or less well marked, though, perhaps, it is only observable when the patient attempts to write (*paralytic form*). A gradually increasing and very decided sense of fatigue is experienced in the hand and forearm, which become, as it were, stiff, and no longer capable of being moved; pain is felt in the whole arm, and, if the act of writing is persisted in, it extends to the shoulder and back. As soon as the pen is laid down the feeling of weakness and exhaustion disappears, to reappear as soon as it is taken up again. It is generally confined to the flexors, or to the extensors, or to the ulnar border of the forearm, and spreads from this as a centre.

The *motility* of the affected muscles for all the coarser kinds of work usually remains unimpaired. The various movements of which they are capable are performed with normal precision and strength, and no anomaly can be observed even when they are called into full action; occasionally, however, particular muscles, especially the extensors of the fingers and thumbs, present distinct paralytic debility; in rare cases the whole arm is similarly affected, and becomes less capable of discharging its ordinary duties. Hasse, however, observed a case of tremor limited to the right hand which interfered with all the ordinary occupations of the patient.

The execution of other kinds of fine work is often much disturbed; those who suffer from writers' spasm suffer also from impairment of the movements requisite for sewing, piano-forte playing, embroidery, buttoning up the clothes, and similar acts; this is not, however, always the case. If the patient have learned to write with his left hand, the spasm not unfrequently extends to this also, and his pleasure in his new acquisition is quickly turned to disappointment.

The most common, and indeed almost constant *sensory disturbance*, is the painful feeling of fatigue in the affected extremity, which may rise to a high degree of intensity and assume a neuralgic character; the shoulder and back are frequently painful, and some of the spinous processes of the cervical and dorsal vertebræ are not unfrequently sensitive to pressure. The pain may extend even to the occiput, and one of my patients, who was a physician, stated that he frequently suffered from deep-seated frontal pain on the left side (does this afford a clue to the localization of the disease?).

Johann Mueller observed slight shocks, resembling those of electricity, in his own fingers. Many patients complain of formication and numbness in the region supplied by one or other of the nerves of the forearm. The existence of true anæsthesia, however, can seldom be demonstrated; the same may be said of hyperæsthesia, and it is only now and then, where neuritis or some other form of inflammation is present, that the presence of a kind of pressure points has been ascertained (see a case described by Runge).

Many other forms of nervous disturbance are usually associated with writers' cramp. These are particularly common in so-called nervous persons, and those who are members of neuropathic families, and who exhibit different morbid phenomena in other regions of the nervous system. These various forms of spasm, strabismus, stammering, spasm of the face, of the throat, etc., are occasionally observed; weakness of the extremities and paraplegic symptoms with tremors do also occur, and very commonly there is great psychical excitability with inclination to mental depression; and it is worthy of notice that in almost all patients of this kind psychical influences, emotions of all kinds, and over-exertions of either the mind or the body, exert a decidedly injurious action on the progress of the disease.

The behavior of the affected muscles and of the nerves supplying them under electrical excitation has been the subject of much investigation, but the results have been almost always negative—that is to say, the muscles have reacted normally; electricity, therefore, affords us no assistance, as regards either the diagnosis or the prognosis of the disease. The presence of slight quantitative alterations, either of increase or of diminution of the excitability, may often be demonstrated by examination with faradic currents. A few observers, as A. Eulenburg, M. Rosenthal, and others, have demonstrated qualitative anomalies from experiments with galvanic currents; these, however, cannot as yet be turned to practical account. Various "business neuroses," which are altogether analogous to writers' spasm in their nature and in their symptoms, require here only to be shortly noticed.

Piano-forte players' spasm is of no uncommon occurrence, particularly in professional players (women), and presents the same features as writers' spasm, such as spasmodic contraction of particular fingers, painful exhaustion and stiffness of one or the other hand, and pain in the shoulder and back, which is most frequently experienced between the scapula and spinal column.

Violin players' spasm sometimes occurs in the left, sometimes in the right hand, either in the form of painful exhaustion and stiffness, or as convulsive spasm of some of the muscles of the head, arm, or shoulder. It renders playing impossible.

Very similar symptoms are presented by *tailors'* and *shoemakers' spasm* (*sewing spasm*). As soon as the patient begins to work, tonic and clonic spasms or functional debility of the muscles of the hand and arm are experienced; but the cases of tetany, which have been often described under the name of "shoemakers' spasm," must be distinguished from this (see the chapter on Tetany).

Smiths' spasm has been occasionally described as a tonic contraction of the muscles of the forearm appearing as soon as the hammer is seized, or as a painful spasm of the muscles of the

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upper arm and shoulder, which are especially called into play in the act of hammering.

Basedow was the first to describe a similar affection affecting milkmaids, under the term *milkers' spasm*, which consists of a tonic contraction of the flexors and extensors of the forearm on any attempt to milk being made, whilst all other occupations can be undertaken with facility.

The number of these forms of spasms could easily be augmented, as analogous conditions occur in all avocations, and they have already been occasionally observed in painters, makers of artificial flowers, harp-players, watch-makers, and turners.

The *course* of all these forms of disease is almost the same. They commence very gradually, but after a time increase more and more rapidly, and it is only in rare instances that they are observed to begin almost suddenly, after some powerful exciting cause, as over-exertion or violent psychical influence.

The symptoms vary greatly in intensity, partly in consequence of external and partly of internal conditions. The patient is rendered worse by bodily or mental exertion, or by strong emotions, whilst rest, discontinuance of the particular occupation which has led to the spasm, and general tonic and strengthening measures, improve his condition.

The *duration* of the disease is usually very protracted, often lasting throughout life. Complete recovery, or even marked improvement, is of very rare occurrence. Arrest of the progress of the disease is more common, and is relatively favorable.

Etiology.—Writers' spasm is met with most frequently in men, much more rarely in women, and this is apparently the result of the different amount of writing required of the two sexes. Pianoforte players' spasm occurs more frequently in women, and particularly in neuropathic persons, who belong to "nervous" families.

The principal cause of the disease is excessive writing (or pianoforte playing, sewing, etc.). It is consequently most frequently observed in writers, secretaries, clerks, merchants and *savants;* but it often occurs also in those who write but little, and who think they have done wonders when they have signed a score of business letters.

The spasm is occasionally and more rarely seen to arise from exposure to cold, after injuries of nerve or muscle, from foreign bodies in the fingers (Hubert-Valleroux), from reflex action consequent on periostitis of the external condyle of the humerus (Runge). In some few cases, neuritis of one or other of the nerve trunks of the forearm has been found to be the cause of the symptoms (Remak, M. Meyer). Such cases, however, do not strictly belong to these forms of spasm, and the same is true of those disturbances in writing which depend on centric (cerebral and spinal) disease.

It is obvious that inconvenient tables, a bad position in writing, tight sleeves, bad pens, and especially hard and pointed steel pens, must favor the development of the disease, since all these means increase the demands made upon the functional activity of the muscles and nerves employed in writing. It is certain, however, that steel pens are not exclusively to blame in producing this form of spasm, since it was known before they were invented, and occurs in those who use only quill pens.

In regard to the *essential nature* of writers' spasm there is still much obscurity, since pathological anatomy has been able to add but little to our knowledge; we are consequently compelled to rely exclusively on hypotheses, and we have indicated in our introductory remarks in what direction they point. The numerous cases that occur cannot, of course, be all comprehended in one category, and it is certain we have to deal with several forms of primary disease. The view that there is an increased excitability, and at the same time great debility of the co-ordinating apparatus (or of certain parts of it), sufficiently accounting for the phenomenon of the spasm with the consecutive exhaustion, in typical cases, and especially in the spastic forms, is highly attractive. At all events, there are here various delicate impalpable trophic disturbances, in regard to the anatomical seat of which we are completely ignorant.

It is very generally believed that there is debility or paralysis of certain muscles, and secondary spasm of their antagonists (Zuradelli, Haupt), an opinion which does not seem to us to be correct for a majority of cases. The hypothesis, that in writers' spasm there is a reflex spasm proceeding from the sensory cutaneous, or sensory muscle nerves (Fritz), seems to us equally ill founded; though it cannot be denied that particular cases originate in a reflex mode; even in these cases, however, it is probable that there is coincident disease in the central reflex apparatus.

Disturbances in the motor paths (peripheral or central), or in the muscles themselves, are very seldom the cause of true writers' cramp; such a connection can only be conceived where actual experiment shows that there is a distinct diminution of the motility, or a considerable diminution of the electrical excitability.

In the present state of our knowledge we are justified in placing the seat of the cause of the typical forms of writers' spasm in the central nervous system, although we are not in a position to locate it with precision. Whether the trophic disturbance is to be sought for in the gray substance of the cervical portion of the spinal cord, or in the cerebral peduncles, or, lastly, in the gray substance of the brain, can only be determined by future investigation.

The diagnosis of this form of the disease is, for the most part, easy. Care must be taken not to confound it with other diseases which may also disturb the handwriting, though they do not belong to the same category. It will be sufficient to refer here to the various forms of tremor, to chorea, to progressive muscular atrophy, and to arthritic disease, to indicate, on the one hand, the most likely diagnostic errors, and, on the other, to enable the physician to avoid them. It is especially important to pay attention to the often almost imperceptible commencement of spinal and cerebral paralyses, for they not unfrequently first appear in the form of disturbances of the more delicate movements, and especially of the writing, though with a moderate amount of attention their true significance will soon be recognized.

It often costs much trouble to determine with precision the particular muscles that are affected and the nature of the disturbance; it is necessary that the examination should be very exact and complete; the patient should be observed whilst he is in the act of writing. Each muscle should be tested, and the characters of his handwriting be studied. Particular attention must also be paid to the determination of the primary disturbing causes, and it may be regarded as a stroke of good fortune if we discover one in an attack of neuritis or in the presence of a painful scar or other reflex irritation, or in the use of bad writing materials.

The *prognosis* of writers' cramp (and the cognate forms of disease) is unfavorable. In all cases it is at least doubtful, and in the majority decidedly unfavorable. Complete recovery is very rare; considerable improvement or arrest at a certain point being more frequent. In a large number of cases no treatment proves of any value, the disease making steady progress and at length rendering writing impossible. Of course the cases in which, when the practice of writing is discontinued, the spasm ceases cannot be included in those that are benefited by treatment. It is obvious that the affection may prove extremely important in its effect upon the support and social position of those who have much writing to do, and such patients are often obliged to take up some other calling. Writers' spasm, as a rule, has no influence on the general health or on the duration of life.

The *treatment* should consist, primarily and essentially, in attention to and removal of the cause, and nothing should be left untried that gives the least prospect of success. The discontinuance of all writing, piano-forte playing, sewing, or whatever else may have led to the occurrence of the disease, or at least the limitation of such occupations to the greatest possible extent, is of the greatest importance. In recent and slight cases this alone will often effect a cure in the course of one or two months. In more chronic cases, however, this abstinence from writing must be insisted on with the utmost rigor, though, unfortunately, the circumstances of the patient render it sometimes impracticable. In cases that are at all severe or obstinate, there is little chance of recovery unless the patient can give up his occupation for six months or a year. If this cannot be done, we must endeavor to assist the patient by recommending the use of good soft pens and suitably made penholders (and I may observe that I have found thick cork holders very serviceable), and by improvement in the method of writing.

Electricity is undoubtedly the most important and effective of the direct therapeutic agents in these affections, and excellent results may be obtained from its use, especially in recent and slight cases. In very chronic cases it is useless, though its failure may in some instances be attributed to the employment of a wrong method.

The *faradic* (or *interrupted*) *current* appears to have little effect, and only proves useful in cases of local anæsthesia or hyperæsthesia, or of paralysis of particular muscles. A few successful cases have, however, been reported (as those by M. Meyer and Zuradelli). Local faradisation of the nerves and muscles, or cutaneous faradisation by means of the electric brush, may be tried in different instances. The purely spastic forms, so far from being improved, are sometimes made worse by the application of faradic electricity.

The *galvanic current*, on the other hand, much more frequently gives decidedly favorable results, as is sufficiently evidenced by the striking improvement in the handwriting that may be observed to occur and to remain for some time during or after the application of the constant current. I have thoroughly satisfied myself of its efficacy, though I have not been able to establish the superiority of any one method of applying it over the others. When good effects have followed, the same results were obtained from all modes of applying it to the arm and neck.

It may be reasonably held, however, that the same method of treatment will not prove equally effective in all cases, and that as a rule different methods must be tried, and tried with perseverance. Electro-therapeutists are by no means in accordance in regard to the most advantageous method in which electricity should be applied. According to the present state of our information it would appear most advisable to galvanize the vertebral column in the cervical region, with ascending stabile and labile currents, and to combine with this the peripheral galvanization of the nerves and muscles of the arm which are especially affected. I believe I have obtained favorable results in several cases by the transmission of galvanic currents, both transversely and in an antero-posterior direction, through the head. Many modifications of this method based on general electro-therapeutic principles, must be adopted in particular cases, but, generally speaking, both the pain and the feeling of fatigue rapidly disappear during the application of these currents, whilst the improvement in writing only occurs after the lapse of some time, and sometimes altogether fails to take place. The galvanic treatment should in all instances be continued for a considerable period, at the very least for several months. The application of the current, which should not be too strong, may be made as often as from three to six times per week.

In cases where the disease is due to neuritis, or other similar conditions, the galvanic current is also the principal remedy; many of my patients have experienced material improvement from wearing a single galvanic element on the arm for a longer period, as, for instance, for several hours daily, or even permanently. (According to Ciniselli's plan, a single plate of zinc and one of copper united by a wire netting covered with silk may be placed on any part of the body, with a moist linen rag beneath.) In cases where the electric treatment proves ineffective, little benefit can be expected from other remedies; nevertheless several additional means may be resorted to, and amongst these gymnastics, shampooing, the employment of tonics and general corroborants, mountain travelling, and a moderate cold water cure may be mentioned. Little can be expected from nervines or antispasmodics, or from narcotics, though they are often enough prescribed in despair on the failure of other means. Strychnia has found its panegyrists, and Rossander cured one case by a combination of shampooing and subcutaneous injections of strychnia. Baths, counter-irritants, spirituous and narcotic embrocations, and liniments almost always fail in producing any benefit.

In desperate cases many patients resort to *mechanical means*, though seldom with success. The simplest measure that can be adopted is to insert the pen into a cork or a thick piece of wood, or to fasten it by means of a ring to the first or middle finger. Attempts have sometimes been made to counteract particular spasmodic movements by means of complicated apparatus, but in most instances without success. Such measures are especially futile in the paralytic form. Many patients are relieved by the method suggested by Tuppert, of applying a narrow bandage or strip of court plaster firmly around the wrist.

Tenotomy of the affected muscles has been recommended as a *dernier ressort*, and Stromeyer having obtained a successful result in one case, by section of the tendon of the flexor longus pollicis, the same plan was tried by Dieffenbach, Langenbeck and others, but for the most part without effect. Tuppert has gone so far as to perform this operation no less than 50 (!) times in one arm, without any better result than "improvement." My own opinion is that tenotomy is absolutely useless in the great majority of cases, and that it is only permissible when the spasm is limited to a particular muscle.

The treatment of the other "business neuroses" above enumerated must be conducted on essentially similar principles.

f. *Spasm of the Muscles supplied by the Lumbar and Sacral Nerves.* (*Plexus Lumbalis and Sacralis*).

Romberg, l. c.—*A. J. Jobert*, de la contract. rhythm. muscul. involont. Clin. Europ. 1859, No. 17.—*Remak*, über Spasm. alternans transvers. Berl. klin. Wochenschr. 1864, No. 10.—*Erb*, Galvanoth. Mittheil. Arch. f. klin. Med. III. p. 350.—*Beitter*, Contractur d. Adductoren beider Oberschenkel. Ztschr. f. Chir u. Geburtk. 1868. —*Kussmaul*, über rheumat. ton. Krämpfe mit Albuminurie, etc. Berl. klin. Woch. 1871, Nos. 42—44.—*Dostels*, Contracture des extrémités infér. etc. Journ. de méd. d. Brux. 1872. Dec.—*Duchenne*, Impotence fonctionn. et spasm. fonct. du long péronier latéral. Arch. gén. 1872.—Electris. local. II. éd. p. 1008.—*Weir Mitchell*, on certain painful affections of the feet, Philad. Med. Times, 1872, Nov.

Isolated and independent spasms affecting particular muscles of the lower extremities are of extremely rare occurrence. It is alike uncommon in practice and in the literature of medicine. As a rule, it is either a symptom of some more general form of spasm, as tetany, tetanus, hysteria, chorea, or epilepsy, and is described under the heads of these diseases, or, and this is most frequently the case, it is a symptom of some central (and especially spinal) disease, to the account of which the reader may refer.

A few observations upon these points will here be sufficient, since, in regard to the etiology and treatment of the several forms, we can only refer to what has been already said in treating of spasms in general and of those of the upper extremity in particular.

Stromeyer was the first to describe the so-called "*spastic contracture of the hip*," and to suggest the means of establishing its diagnosis. By this term is signified a tonic spasm of the iliac and psoas muscles and of the quadratus lumborum and occasionally of one or two of the muscles of the front of the thigh. The thigh is kept in a strongly flexed position, the tendon and muscular belly of the ilio-psoas muscle project strongly, the pelvis appears to be raised on the affected side, the limb is shortened, and in walking the patient inclines to this side. Passive extension cannot be performed, and any attempt to straighten the joint causes acute pain in the tense muscles, and frequently in the knee also. This form of spasm is most frequently induced *directly* by disease of the lumbar vertebræ, psoitis, and psoas abscess, and in a reflex manner by diseases of the hip joint (coxitis and articular neurosis).

Spasm of the *quadriceps extensor femoris* is on the whole of rare occurrence. Rigid extension of the knee joint, such as is seen in tetany and in articular neuralgia of this joint, results from tonic spasm. I have seen clonic convulsion of this muscle, in a case of articular hyperæsthesia, whenever the patella was touched. A. Eulenburg, in his treatise on nervous diseases (p. 700), mentions a case of clonic spasm of the right quadriceps which was induced by every attempt to walk or stand, and which was cured by electricity.

Contracture of the *adductors* of both thighs was observed by Beitter, which probably resulted from rheumatic inflammation of both hip joints. It has also been seen as a symptom of tetany by Stich.

Remak has described a remarkable case of spasm of the *glutæi muscles*. It consisted of a rhythmical and synchronous spasm in the muscles of the right arm and left leg, alternating with a similar spasm in the left arm and right leg. The spasm in the legs consisted of a series of contractions of the glutæi, in consequence of which the leg was drawn backwards in walking, and fixed in that position.

Spasm of the *flexors of the leg* (biceps femoris, semi-tendinosus and semi-membranosus) is not unfrequently observed as a stiff contracture, in hysterical patients or in those suffering from disease of the spinal cord or from disease of the knee joint. The result is that the knee joint is kept in a strongly flexed position, which may proceed to such an extent that the heel and the buttock are brought into contact. In such cases active extension is impracticable, and passive extension is extremely difficult and painful.

Spasms of the *anterior muscles of the leg* (supplied by the peroneal nerve) are upon the whole of rare occurrence. I have, however, seen one such case, in which there was contracture of all the muscles supplied by the peroneal, in consequence of paralysis of the muscles supplied by the tibial nerve (club-foot). Weir Mitchell has described a peculiar and painful contracture of the tibialis anticus, peroneus longus and gastrocnemius, which occurs in young people after long standing, and leads to deformity of the feet. Duchenne enters very fully into the question of the influence of spasm of the peroneus longus on the origin of certain forms of club foot, and draws a distinction between functional spasms, which only occur when the leg is brought into use, and persistent contracture of the muscles; the two can easily be distinguished from one another, but the details belong to surgery. Jobert de Lamballe reports a remarkable case of rhythmic spasmodic contraction of the peroneus brevis, which was associated with an audible sound, when the tendon of the contracted muscle returned on relaxation to its normal position; this spasm resulted from exposure to cold, and first appeared in the right and subsequently in the left foot; it was ultimately cured by tenotomy. (This calls to mind the knocking spirit which excited so much attention in the Palatinate, and which Schiff exposed, by demonstrating the existence of a similar mechanism under the influence of the will.)

Spasm of the *muscles of the calf of the leg* (supplied by the tibial nerve). This belongs to the more frequent forms of spasms; it is commonly known under the name of "cramp in the calf," affecting the gastrocnemius (see the following section), and it is also a very frequent phenomenon in tetany (see Tetany). Contractures of the sural muscles are of very common occurrence, as a consequence of paralysis of the peroneal nerve, of joint disease, and of diseases of the spinal cord, and cause "pes-equinus," the heel being strongly elevated, the point of the foot depressed, and the toes flexed. Cramp in the calf of the leg is not unfrequently seen in sciatica, either owing to direct or to reflex irritation of the nerves. I have also seen reflex contractions occur in the calf, in hyperæsthesia of the knee-joint.

More or less diffused spasm of the whole inferior extremity, or of both extremities together, occurs occasionally in hysteria. I have seen strong clonic spasms of the whole of the left leg in an epileptic patient; the spasms occurred frequently, though not always, as a kind of motor aura. Tonic and clonic spasms of the lower extremities are very common in various diseases of the spinal cord, and may for the most part be regarded as exalted reflex action; they appear as tonic flexion or extension of the extremities, and frequently also in the form of violent clonic tremblings in certain positions, as in passive dorsal flexion of the foot.

An affection of both lower extremities, which appeared after long antecedent strain of the right leg, has been described by Kussmaul as a peculiar form of rheumatic tonic spasm. The chief symptoms were a strong and painful extension of the whole leg and foot with flexion of the toes. It lasted for seven days. From time to time the muscles of the neck and back, and, transiently, those of the right shoulder became rigid; albumen was present in the urine, and there was free perspiration. Kussmaul considered this affection to be an intermittent rheumatic rigidity of the muscles, distinct from tetany. A case reported by Dostels probably belongs to the same category; it was that of a girl, aged nine, in whom, after over-exertion, violent spasms of the calves occurred, which gradually developed into permanent contracture of both legs, though undergoing paroxysmal augmentations of intensity; recovery took place after the affection had lasted for three days. These cases serve to indicate that these rheumatic forms of spasm have their real seat within the spinal canal, and it is very desirable that all cases of this kind should be reported.[1]

The *treatment* of the above-mentioned forms of spasm must be conducted on the principles already repeatedly given—it is unnecessary to enter into details. The causal indications must be attended to; faradic and galvanic currents are to be applied in the ordinary way; nervines and anti-spasmodics should be prescribed, and, lastily, tenotomy and orthopœdic measures may be practised, according to the nature and peculiarities of the case.

Kussmaul's case recovered after the application of cupping to the spine and the administration of confection of senna and Vichy water; the case of Dostels, after the use of vapor-baths and quinine. (The reader is referred to the treatment of Tetany.)

g. *Cramps* (*Crampi*).

Hasse, Nervenkrankheiten, 2. Aufl., p. 161.—*Griesinger*, Infections-krankheiten, 2. Aufl., p. 421.

By the term cramps is understood a transient tonic spasm, occurring in different parts of the body, and generally confined to a single muscle; it is very painful, and in general lasts only for a few minutes, though it may recur rather frequently; occasionally, as in cholera, but seldom otherwise, it constitutes a very troublesome and unpleasant symptom.

A good type of this form of spasm is familiar to every one in "cramp of the calf of the leg," which is a sudden and very painful contraction of the muscles of the calf, occurring in consequence of some incautious movement, or even spontaneously, during sleep. The gastrocnemius swells up and is as hard as a board; its contour is sharply defined, yet the tendo Achillis is very little shortened, and the heel is very slightly raised. The pain is severe, and the contracted muscle is very sensitive to pressure, friction or passive extension. In the course of a few seconds, or minutes, the spasm, and with it the pain, ceases;

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[1] The remarkable forms of spasm chiefly confined to the lower extremities, described by Bamberger and Guttmann, under the name of "leaping spasms," as well as those occurring in the feet, and those which are observed in what has lately been described by Hammond as "athetosis," are of centric origin, and therefore do not belong here.

but an unpleasant sensation of fatigue, with augmented sensitiveness to pressure, often persists for some time. In rare cases, ruptures of small blood-vessels take place during the cramp, and ecchymoses and painful swellings form, which only slowly disappear. Occasionally the spasm recurs, and it may even do so frequently for hours together, when it becomes a source of torment to the patient, and deprives him of sleep.

The spasm presents similar characters when it attacks other muscles, as the small muscles of the sole of the foot, the extensor pollicis, the extensor longus digitorum, the adductors of the thigh, the latissimus dorsi, the sacro-lumbalis, the extensors of the wrist, the platysma, the sterno-cleido-mastoid, the trapezius, and the rotatores capitis, etc.

Cramps assume their highest importance in Asiatic cholera (and in severe cases of cholera nostras); they supervene during the choleraic attack proper, first and most frequently in the calf of the leg, then in the thighs and toes, the arms and fingers, and less frequently in the muscles of the face and abdomen. Such cramps are extremely painful, lasting for some minutes, then ceasing, and then again recurring, to the great torment of the patient. According to Schultz, the reaction of the affected muscles to electric stimuli is increased, but the application of the current is very painful.

Etiology.—The most frequent causes of cramp are fatigue and over-exertion, and nothing is more common than for cramp of the leg to occur after mountain climbing, after a night devoted to dancing, or after prolonged swimming. It then comes on either quite spontaneously, as, for example, in the night, or when certain movements are with much exertion made. Cramps are indeed easily brought on by violent and sudden movements made in an awkward position. They occur in many patients upon comparatively slight mental exertion, and many elderly persons have a predisposition to cramps. Certain muscles, again, may be particularly liable to be affected, and this is especially the case with those which have been already attacked with cramp.

It is doubtful whether pressure on the nerve trunks and muscles is capable of producing cramp, and the statements sometimes made, that disturbances of the circulation, nervous stasis, phlebectasis, etc., readily occasion cramp, does not appear to be satisfactorily established, nor does it appear to be quite certain that it can, as is usually admitted, be caused by reflex irritation (intestinal disease, diarrhœa, and worms). It is especially in cholera that reflex irritation proceeding from the intestine appears to be insufficient to explain the occurrence of cramps, and it is much more probable that the real cause lies in the great changes in the constitution of the blood, which lead to disturbances in the nutrition of the muscles; thus it may be either the dryness of the muscles, caused by the great transudation of water, the arrest of the circulation in the arteries, or the accumulation of excrementitious substances in the muscles, which occasions their peculiar convulsive excitability. I saw in a rapidly progressing case of diabetes, in an old gentleman, obstinate and violent cramp in the calf of the leg, which recurred at intervals throughout the night. Was this due to excessive transudation of water from the blood, or to accumulation of sugar in the blood? Slight cramps may be induced in many persons by strong faradisation of the motor nerves and of the muscles, especially in the muscles of the lower extremity.

The real nature of cramps, and the precise mode in which they are established, are still unknown. To maintain, as Hasse does, that they are due to reflex stimulation, proceeding from the sensory nerves of the muscles, does not appear to us to be satisfactorily established. At all events, it may be admitted that some change occurs in the muscular substance itself, so that there is an increased excitability, and we may further admit that a transient disturbance in the nutrition of the muscles is produced either by great fatigue, by diminution in the normal amount of water, or by disturbances of the arterial circulation, and that this disturbed nutrition manifests itself by augmented excitability, and leads to the production of cramp, on the slightest motor stimulus, whether of a voluntary or of a reflex nature. At the same time, we do not mean to say that similar trophic disturbances and exaltation of excitability may not also be experienced by the motor nerves. When cramp takes place in healthy muscles from violent exertion, it can only be admitted that it is due to augmentation in the intensity of the stimulus.

The *prognosis* of ordinary cramps is generally favorable, but they sometimes prove a very obstinate and frequently recurring affection in old people. The cramps that accompany cholera do not notably influence the prognosis in this disease.

Treatment applied to this form of spasm is usually considered unnecessary. Most people are acquainted with the ordinary domestic remedies by which relief may be obtained ; amongst them may be mentioned complete rest of the muscle, friction and shampooing, and passive extension. In more obstinate cases, warm baths, spirituous, anæsthetic, and narcotic frictions may be tried, and the galvanic current is sometimes serviceable.

Subcutaneous injections of morphia have proved extremely useful in the cramps accompanying cholera, as have also shampooing, embrocations with chloroform liniment, friction with pounded ice, etc.

h. *Tetany.*

Steinheim, zwei seltene Formen von hitzigem Rheumatismus. Hecker's Annal. XVII. 1830.—*Dance*, sur une éspèce de Tétanos intermittent. Arch. génér. 1831. Bd. 26.—*Constant*, sur les contract. essentielles. Gaz. méd. 1832.—*Tessier et Hermel*, de la contracture idiopath. etc. Journ. de méd. 1843.—*Weisse*, ton. Kr. d. Finger und Zehen. Journ. f. Kinderkr. 1844.—*Marotte*, Observ. d. Contract. essent. Journ. de méd. 1845.—*Delpech*, Mém. sur les spasm. musc. idiopath. Paris 1846.—*Trousseau*, Gaz. des hôp. 1845, No. 87 ; 1851, No. 128 ; 1856, No. 72 ; 1860, No. 44 ;—Medic. Klin. d. Hotel Dieu, Deutsch von Culman, II. p. 155. 1868.—*Clemens*, z. Lehre von den Nervensympath (Schusterkramp.) Ztschr. f. rat. Med. X. 1851.—*Lucien Corvisart*, de la tétanie chez l'adulte. Paris, 1852.—*Rabaud*, recherch. sur l'his. etc. des contract. des extrém. Paris 1857.—*Ercole Ferrario*, Gaz. med. ital. Lomb. 1857, No. 36.—*Fil. Lussana*, sulla contrattura reumatica. ibid. 1857, No. 34.—*Hasse*, Nervenkrankheiten, 2. Aufl. 1869.—*Kussmaul*, über rheum. Tetanus etc. mit. Albuminurie. Berl. klin. Woch. 1871, Nos. 41—44.—Ueber Tetanie. ib. 1872. No. 37.—*Bauer*, Trousseau's Tetanie ?—Ergotismus ?—ib. 1872, No. 44.—*Sitch*, 2 Fälle von Schusterkr. Arch, f. klin. Med. XI. p. 528. 1873.—*Erb*, z. Lehre von der Tetanie. Arch. f. Psych. und Nerv. IV. 1873.—*Riegel*, z. Lehre v. d. Tetanie. Arch. f. klin. Med. XII. 1873 (see the same also for a very complete bibliography of the subject.)—*F. Schultze*, über einige Fälle von Tetanie. Berl. klin Woch. 1874, No. 8.

The term *tetany* was given by L. Corvisart to a peculiar and rather common disease, which consists of paroxysms of tonic contractions of certain groups of muscles. It for the most part affects the muscles of the upper extremities, the forearm and hand, and frequently those of both upper and lower extremities, but seldom those of the lower alone; it consists of painful spasms lasting for a variable period, but always intermittent, sometimes recurring, and generally affecting single muscular and nervous regions. Slight sensory disturbances are generally present. The spasms are often associated with certain movements and occupations, but must not be confounded with business neuroses affecting co-ordination. It occurs perhaps still more often quite independently of all muscular effort, attacking the patient by preference at night and on holidays, as several observers have remarked.

The literature of tetany is tolerably rich, and has been very completely collected by Riegel; the affection has been particularly studied and described by French physicians. The first clear description of it was given by Steinheim, in the year 1830. In France, in 1831, Dance commenced the series of publications upon this subject. Unfortunately almost every author has given a special name to the disease, so that a great confusion of terms exists. We shall here give the most important, to assist the student in his reading. Dance described the affection as "tétanos intermittent." Numerous other names were afterwards applied, such as "contracture essentielle" (Constant), "rétractions musculaires spasmodiques" (Murdoch), "spasmes musculaires idiopathiques," "tetanie" (Lucian Corvisart), "tetanille" (Trousseau), "Brachiotonus rheumaticus" (Eisenmann), "Schusterkrampf" (Clemens), "Rheumatische Contractur," "Tonischer Beschäftigungskrampf" (Benedict); and some of the so-called carpopedal spasms belong to the same category. The term "*tetany*," which was introduced by Corvisart, and which has received very general adoption, is the best.

Etiology.—Certain periods of life and stages of development exhibit a decided predisposition to the disease. A rather strong tendency to it is exhibited during early childhood, from the fourth to the sixth year. Next to this is the period of puberty and youth, by far the majority of cases in adults occurring in those who are between sixteen and thirty years of age. Further, the disposition to tetany is considerably increased during pregnancy, in the puerperal state, and in lactation (contracture des nourrices, Trousseau). The occupation of the patient does not, however, exert any great influence in producing it, though the disease has been described as "shoemaker's cramp," and as being associated with certain employments. It appears, however, in artizans of all classes, and its frequent occurrence in young children and women is opposed to this view.

Amongst the *exciting causes*, catching cold is both the most important and the most common, and this statement is supported by the fact that many physicians have regarded it as an exquisite example of rheumatic disease. Working in the wet or cold, or in water, sleeping on the damp ground, have very often been regarded as causes, and the swelling in the joints which occurs in many instances, indicates that this disease has a somewhat close relation to true rheumatism.

The relation of tetany to typhoid fever, measles, cholera, Bright's disease, febris intermittens, exhausting diarrhœa, etc., of which it often constitutes a sequela, is still imperfectly explained. These diseases, in many cases, act as predisposing, but in others as direct causes. The observations of Bauer appear to show that poisoning with secale cornutum (ergotism) is capable of directly inducing tetany; and Moxon has recently called attention to the similarity of the symptoms of tetany and ergotism.

Certain influences, which at present may be termed reflex, appear to be of great importance, though we are unable to give a satisfactory account of their mode of action. Amongst these may be included the pathological stimuli which proceed from certain developmental and physiological processes, as dentition, puberty, pregnancy, and lactation. Suckling appears to be particularly favorable to the occurrence of tetany, and this indeed to so remarkable an extent, that Trousseau was for some time of opinion that the disease was confined to nursing women.

In the next place, irritation of the alimentary canal (all forms of intestinal disease, worms, etc.) is held to be peculiarly effective in inducing tetany, and many observations might be adduced to show that protracted and exhausting diarrhœa, both in children and in adults, is very frequently followed by tetany. The connection of worms with tetany has also been very satisfactorily demonstrated.

Lastly, we must not omit to say that tetany has often been seen to follow various psychical influences, as violent emotional excitement, etc.

Symptoms.—The attack of spasm is usually preceded by certain premonitory symptoms, which are chiefly of a sensory character, such as a peculiar creeping or dragging sensation, formication, feelings of heat and cold, with, sometimes, well-marked pain in the forearm and hand. After a time, slight contractions, or feelings of stiffness, in particular fingers occur, which are especially noticed when any object is grasped; and these are more or less quickly succeeded by the spasm in its full intensity, which frequently occurs after the performance of certain movements, or on strong exertion. It consists of a series of separate attacks.

Each attack generally commences with a rigidity, swiftly affecting the hands and fingers, which are usually flexed, so that the hand presents a peculiar, conical form, exactly resembling that which is produced when the ulnar nerve is powerfully stimulated by faradic currents; the thumb is strongly adducted, the two borders of the hand are approximated to each other; the second, third, and fourth fingers are strongly, whilst the first is only slightly flexed, and all are firmly applied to the thumb. Coincidentally with this the wrist is strongly flexed towards the ulnar side. In other instances the fingers are all firmly closed and rigid, so that the hand resembles a paw; or, the region of distribution of the median nerve may be chiefly affected, in which case the thumb is turned inwards, and tightly enclosed by the spasmodically bent fingers; in rare instances the hand is violently flexed backwards; the forearms are then semi-flexed, the upper arms are strongly adducted, and the forearms are thus crossed upon the epigastrium. Acute pain is experienced in the rigid muscles, which are tightly stretched, and feel hard; their tension undergoes considerable variation during the attack, though they never entirely become relaxed. They offer considerable resistance to passive extension, and when force ceases to be applied they generally return to the position assumed in the spasmodic state. In many instances the spasm affects the lower extremities also, compelling them to assume a rigid and extended position; the knee is straightened, the head is drawn strongly upwards, and the toes are strongly flexed. The patient is unable to walk, and experiences great pain and hyperæsthesia of the muscles. He cannot perform any kind of work. Spasms, causing flexion of various joints, likewise occur in the lower extremities.

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It is only in very severe attacks that the muscles of the back and neck are affected, those of the abdomen and diaphragm still less frequently so; whilst, most rarely of all, the muscles of expression of the face, and those of the tongue, and of mastication, are involved. When, however, these are affected, the attack is of a very threatening character, tonic spasm of the diaphragm being especially and immediately dangerous to life.

The spasm may last for a few minutes, a quarter of an hour, or even for some hours, though seldom longer than twelve hours; its violence then gradually abates, a rigidity and immobility of the muscles, with a painful sensation of fatigue, remaining for some time. After an interval of longer or shorter duration, a fresh attack comes on. Days, or even weeks, may intervene between two attacks, but, as a rule, they succeed each other after an interval of a few hours, recurring several times in the course of the day. It is only in very severe cases that they follow one another so rapidly that the patient remains but a few minutes free from spasms.

The affection consists of a greater or less number of such attacks, which may only last for a period of several days or weeks, but generally they continue for several months. In the intervals the patient generally feels in perfect health, is able to walk about and pursue his ordinary avocations; at the same time he experiences a certain degree of weakness and incapacity for exertion in the muscles of the limbs, and more rarely there is some disturbance of the general health, indicated by feverishness, furred tongue, etc. The attacks supervene without any obvious exciting cause, sometimes in the morning, as soon as work commences;

sometimes in the evening, when exhaustion is felt. Their occurrence is promoted by the free use of spirituous liquors. Occasionally, but very rarely, they result from reflex irritation of the skin. Trousseau has drawn attention to a remarkable, and, it would appear, very characteristic symptom, which shows that the disease still exists even during the intervals. If the larger arterial or nervous trunks of the upper extremities be compressed, a well-marked attack of tetany may be induced in the course of one or two minutes, which lasts as long as the pressure is maintained. Kussmaul observed, in one case, that the attack only came on when pressure was made on the arteries, but not when this pressure was applied to the veins. It is much more difficult to demonstrate "Trousseau's symptom" in the lower extremity by pressure on the femoral artery, or sciatic nerve. As long as the disease is not completely cured, this symptom may be shown to exist in many cases, and constitutes an important aid to diagnosis.

Compared with the *motor symptoms*, the *concomitant symptoms* of the disease are comparatively trivial. In slight cases, which sometimes occur in robust young persons, laborers, and the like, they may be entirely absent. Well-marked symptoms, however, are usually present, especially sensory ones; thus, in addition to pain in the muscles, a tearing sensation is experienced in the course of the nerve trunks, which may extend to the shoulder and hip. The attacks are preceded and accompanied by creeping sensations, formication, and other abnormal feelings. True anæsthesia of the skin has only been observed in a few cases. One or two instances of muscular anæsthesia have been reported. Redness and œdematous swelling of the skin around the joints, congestion of the head, headache, giddiness, and humming sounds in the ear, are of rare occurrence. Disturbance of the general health is also rare, though in severe cases there is a certain amount of fever present. Increase of temperature has only been noted in a few cases (Stich), but augmented frequency of pulse, loss of appetite, and coated tongue, are of more frequent occurrence. Free perspiration is of not uncommon occurrence during the acme of the attack. Respiration may be interfered with by spasm of the diaphragm, and of other respiratory muscles. The true cerebral functions are almost always unimpaired. The disease may of course be complicated by all possible symptoms belonging to the primary disease.

Electrical Relations.—The behavior of the motor nerves under the influence of electric currents, appears in tetany to be of special importance. By several observers, as by Benedict and Kussmaul, an increased excitability has been affirmed to exist. In two cases I have repeatedly investigated, by the most exact methods at my command, the conditions of electric excitability, with the following results: there was increased excitability to faradic currents in all the easily accessible motor nerves of the trunk, but *not* in the branches of the facial nerve. There was also very considerable increase of the galvanic excitability in all the motor nerves of the body, with the exception of the facial. The increase of galvanic excitability was recognized (in addition to the disproportion between the amounts of the motor and sensory reactions) by the early appearance of cathodal-closing contractions, by the appearance of cathodal tetanus with currents of uncommonly small strength, and, lastly, by the occurrence—which has not been hitherto observed in man—of anodal-opening tetanus with currents of very moderate strength. The greatest increase of excitability coincided with the time of the best-marked and most frequent attacks of tetany, and there was a decrease in the excitability as they became less frequent; and, finally, when the patient had completely recovered, the electric excitability was found to be approximately normal. A distinct parallelism could thus be demonstrated to exist between the occurrence of spasms and the increase of electrical excitability, suggesting a causal relation between the two phenomena, and that the symptoms observed in tetany are consequent on some primary nervous disturbance.[1]

The essential nature of tetany, it is obvious, cannot be sought for in coarse anatomical and pathological changes, as is sufficiently shown by the complete freedom of the motor apparatus in the intervals of the attacks. Finer disturbances of nutrition must evidently be present, to which the remarkable attacks of spasm are due. The proof of the existence of such delicate trophic disturbances, in the peripheric motor nerves, must be furnished, as it appears to us, by the results of electrical examination ; the great increase of excitability cannot be due, we think, to any other cause than the molecular changes of the nerve substance. It appears then that we do not go too far in attributing the essence of tetany to delicate trophic disturbances, occasioning great increase in the excitability of the motor apparatus, and we should expect spasmodic attacks to occur whenever any unusually strong stimulus affects the motor nerves. Such stimuli may proceed from voluntary efforts to perform various muscular movements (occupation-spasm), psychical excitation, reflex excitation, such as suckling, intestinal diseases, worms, etc. The periodic recurrence of spasm, and its occurrence at various times and under different circumstances, are perhaps attributable to varying conditions of nutrition and excitability in the nerves, to fatigue and exhaustion, though our information in regard to all the minor details of the process is still very imperfect.

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[1] It is probable that exact electrical investigation, as well as Trousseau's symptom, would afford a means of recognizing a latent condition of the disease, or that condition in which no attacks of tetany are present, though the disease is not entirely cured. The persistent increase of faradic, and especially of galvanic excitability, would constitute the characteristic symptoms of this condition.

The question now arises how far this increase of excitability affects the motor nerves. It is very improbable that it should be confined to the peripheric nerves, and indeed everything tends to show that the central parts of the nerves are also implicated, and that tetany is really a centric affection. The coincident affection of so many groups of muscles, their symmetrical affection on both sides of the body, the sensory disturbances that are associated with the motor symptoms, the great resemblance of the whole disease to tetanus, are all points in favor of its centric origin. In favor of this also is the remarkable contrast presented in one of my cases between the nerves of the trunk and the facial nerve in regard to the increase of excitability. It does not therefore appear to be improbable that the primary trophic disturbance in tetany extends upon the spinal cord, and that in the majority of cases it is extremely delicate and impalpable. It is at present impossible to determine with any precision whether the disease of the spinal cord is primary and the trophic disturbance (that is to say, the increase of excitability) of the peripheral nerves is only secondary, or whether it is a disease extending uniformly along the peripheral and spinal motor nerves.

Kussmaul also argues in favor of the central seat of this disease, supporting his views by the occurrence of slight retinitis in the case observed by him, together with the rigid contraction of the muscles of the calf, and debility (resembling paralysis) in the lower extremities, after the disappearance of the tetany. He also mentions that he discovered the presence of myelitis in a young man, who suffered from paralysis of the legs a few weeks after recovery from severe tetany. The slight lesions that have been discovered in the post-mortem examinations that have been made in the few fatal cases that have occurred, serve to confirm this opinion. Trousseau found hyperæmia of the meninges and softening in the upper part of the spinal cord. Ferrario found traces of inflammation of the spinal cord and of its membranes in several cases, but it must always be borne in mind that it is only the most severe cases that terminate fatally, and that great difficulty is experienced in demonstrating pathological changes in the spinal cord. We must leave the question open for further investigation.

The *course* of tetany varies extremely in different instances, and is for the most part irregular; the attacks are sometimes more, sometimes less frequent; it often remains for a long period latent, and then from some inducing cause again breaks out. All possible transitional forms may be met with, from the slightest to the most severe, in which the most violent attacks follow one another with great rapidity. According to Trousseau, three degrees of the disease may be recognized, though it is impossible to define the limits of each very exactly: in the first degree the spasm is limited to the extremities, the attacks are not very frequent, and there are no general symptoms. In the second degree the muscles of the trunk also are in part affected, the attacks are more frequent and violent, and there are some indications of the disturbance of the general health, such as debility, fever, loss of appetite, profuse sweating, etc. In the third degree the facial and masticatory muscles, the muscles of respiration and the diaphragm participate in the spasm, the attacks assume a very threatening character, follow each other stormily, and the patient becomes extremely exhausted; in its aspect the disease resembles tetanus.

The disease is seldom of short *duration;* it very rarely terminates in the course of a few days; in most instances weeks and months elapse before the disease completely ceases; the period of latency, in particular, may last for a long time, and apparent relapses are certainly often only indicative of the fact that the disease had not entirely run its course. Trousseau's symptom affords us valuable information in this direction, and so too does careful electrical investigation.

The *termination* of the disease is usually favorable, complete recovery taking place in by far the majority of cases. In a few instances paresis or paralysis of the extremities persists for some time; death is very rarely caused by tetany, though it may occasionally result from spasm of the diaphragm, general exhaustion, or consecutive spinal disease. Such an event is particularly to be feared in children who have been reduced by diarrhœa.

The *prognosis* of the disease is consequently sufficiently simple; in most instances it is very favorable, but it becomes unfavorable when the attack is very severe, when the malady has been of long duration, when there are evident signs of serious centric disease, when the patient is very young, or is of a bad constitutio and when the primary disease is of a serious character.

The characteristic grouping of the symptoms renders *the diagnosis* comparatively easy; the seat and the distribution of the tonic spasm, the implication of certain nerve regions, the characteristic position of the extremities, the intermitting character of the attacks, the existence of Trousseau's symptom, and the absence of cerebral disturbance, are circumstances which collectively prevent any doubt in regard to the diagnosis of tetany. The most severe cases alone, in which trismus and a high degree of spasm of the muscles of the trunk are present, resemble tetanus; but even here the whole course and the complete intermission of the attacks, the commencement of the spasm in the extremities, and their centripetal extension, in consequence of which the masticatory muscles are ultimately attacked, the absence of exalted reflex excitability, the presence of Trousseau's symptom, etc., enable us readily to diagnosticate tetany.

The *treatment*, in accordance with the great diversity in the character of the attacks, is extremely varied. In the first place, the causal indications may require very different measures to be adopted; in recent cases, caused by catching cold, diaphoretics and anti-rheumatics may be tried; in other cases intestinal diseases will have to be subdued, worms removed, and conditions of marasmus and inanition combated; tonic and nerve-strengthening treatment is frequently requisite, and must be pursued in accordance with the principles and the means which have been already several times mentioned.

Very various measures have been adopted, and sometimes with manifest success, for the relief of the spasms themselves. The most prominent of these are the narcotics and anæsthetics. Chloroform has in particular had many advocates, some recommending that it should be inhaled (even to narcosis, Grisolle), some that it should be administered internally, and some that it should be applied externally in the form of liniments or fomentations; it appears, when energetically employed, to exert a powerful influence in reducing the severity of the spasm, though certainly not in all cases. Ether has been used in a similar manner. The subcutaneous injection of morphia occupies a very prominent place amongst the narcotic remedies. Trousseau strongly recommended a combination of opium with quinine. English authors praise the hydrate of chloral.

Electricity has up to the present time been employed in only a few cases, though it appears to be well worthy of further trial. Although cases of tetany cured by cutaneous faradisation and by faradisation of the antagonists of the contracted muscles have been reported, greater effectiveness might, even *à priori*, be attributed to the galvanic current, more especially if the view of the centric nature of the disease be confirmed by further research. In the meanwhile, it is very desirable that this method of treatment should be perfected by further study. Stich obtained a favorable result from the application of ascending stabile currents to the affected nerves. I saw recovery take place in one case from the stabile application of the anode (also with an ascending current) to the vertebral column and the nerve trunks chiefly affected. I cannot, however, state positively that the result was due to the treatment. The spasms may in some instances be seen to disappear permanently after one or several applications of the galvanic current.

Good results may generally be anticipated from the employment of *baths* and other hydropathic procedures. The violence of the attack is often mitigated by tepid baths and by warm arm-baths, and the same effect is thought by some (Wilks, Ritchie) to be attained by the application of cold water bandages to the extremities. Rubbing the back with ice proved serviceable in one very severe case. Trousseau states that he obtained some improvement from directing the patients to dip their hands in cold water, or to walk about with bare feet on a cold floor.

Amongst the *nervine remedies*, bromide of potassium appears to be the most deserving of confidence, and large doses (thirty grains, Haddon) may be given at short intervals. Arsenic, valerianate of zinc, castor, assafœtida, etc., may also be prescribed. A large assortment of drugs is, however, rarely required, since the course of the disease is usually favorable.

Neither local nor general bleeding is now adopted, except under very peculiar circumstances; local abstraction of blood and derivation over the vertebral column may sometimes prove serviceable.

The dieting must vary with the circumstances of the case. In all cases the affected limbs should be kept at rest; all work, and especially over-exertion, should be avoided, and neither exposure to cold nor mental emotion allowed, beyond what the strength of the patient can bear.

i. *Contractures.*

Hasse, l. c. p. 316.—*A. Eulenburg*, l. c. pp. 611 u. 637.—*Volkmann*, Pitha-Billroth Handb. d. Chir. II. 2. Abth. p. 727. 1872—Klin. Vorträge, No. 1. 1870.—*Hueter*, z. Aetiol. d. Fusswurzelcontracturen. Langenbeck's Arch. Band IV.—

Benedict, l. c.—*Remak*, Galvanother. 1858.—*Hitzig*, Auffassung einiger Anomal. d. Muskelinnervation, Arch. f. Psych. u. Nerv. III. 1872. See also the treatises on Surgery and Orthopædics.

It is impossible here to avoid making some reference to contractures, although a large proportion of them cannot be regarded as symptoms of spasm, being traceable to a very different origin. The frequency with which they occur, and their manifold relations to nervous spasm and paralysis, justify their being mentioned here, though in most cases they are of a secondary and symptomatic nature, and their practical importance is essentially surgical and orthopædic. The conformity with which contractures, however produced, manifest themselves, renders it judicious to consider them from a general point of view, though some of them are undoubtedly not of nervous origin.

Under the general term, "contracture," is understood *any persistent shortening of the muscles*, by which their point of attachment are permanently approximated, by the forces inherent in the muscles themselves, to a greater extent than is the case when they occupy a middle position, or position of rest. Contractures may be arranged in three large groups: 1. Those which are of *secondary* origin, and are consequent upon other forms of disease, such as paralysis and anchylosis; the so-called *paralytic* contractures form the greater number of this group; 2. Those which arise from *anatomical changes in the muscular tissue itself* (myopathic contractures); 3. Those which are induced by *abnormal innervation*, or *abnormal irritation of the motor nerves* (*neuropathic* contractures).

The first group includes, almost exclusively, cases which arise from paralysis of all kinds; the antagonists of the paralyzed muscles are those affected with the contractures; they undergo a gradually increasing and persistent shortening, which may at first be readily overcome by counter-extension, but ultimately becomes quite fixed and inextensible. Very similar contractures may also occur in completely paralyzed muscles, when favored by purely mechanical conditions, such as those which are frequently observed in the spinal paralysis of children. In both cases, trophic disturbances of the muscles occur, which cannot be demonstrated morphologically, but in consequence of which the muscles lose their capacity of elongating to their normal extent (simple trophic shortening of Volkmann). The muscles, when thus affected in young individuals, are arrested in their growth, and at a later period the fibres undergo fatty degeneration and atrophy, with hypertrophy of the connective tissue and retraction.

The mode of origin of this simple kind of contracture is tolerably simple ; the essential feature it presents is a frequently repeated or persistent mechanical approximation of the points of attachment of the muscles. This is occasioned in two ways : either by the active voluntary contraction of the antagonists of the paralyzed muscles, which shorten themselves with each voluntary effort, the paralyzed muscles then being incapable of again effecting their extension (the shortening thus becomes permanent) ; or by the paralyzed members assuming certain positions in accordance with the law of gravity and the position of their centre of gravity, in consequence of which the points of attachment of certain groups of muscles are approximated; this gradually leads to contracture, and it is in this way especially that the pes equino-varus, so common in the spinal paralysis of children, originates. But even here, in all probability, direct changes occur in the muscles themselves, a kind of connective tissue sclerosis of the muscular tissue, which facilitates the fixation of the contracture. In this way are produced the antagonistic contractures that occur in the various forms of paralysis, especially in traumatic paralyses and in those contractures that are so frequent in the spinal paralysis of children.

Contractures of the same kind arise in a similar manner and from the same conditions (persistent approximation of the points of attachment) in diseases of joints, of the articular extremities of bones, of ligaments, etc. If, in consequence of these diseases, the joints are brought persistently into some position different from their mean position (excessive flexion or extension), part of the muscles will be unnaturally shortened, their points of attachment will be approximated, and after a time a contracture will become established, as may indeed be not unfrequently observed a few weeks or months after the joint has assumed its false position. Lastly, the use or non-use of certain parts on one side of the body only; the permanent maintenance of the vertebral column in an oblique position, etc., may, by causing undue approximation of the points of attachment of the muscles lead to a permanent muscular contracture.

The second group includes *myopathic contractures.* These also may arise in very different modes. The contracture, however, is always due to an anatomical change in the muscular substance itself. As a rule, connective tissue induration (cirrhosis of the muscles) is present with coincident atrophy and partial fatty degeneration of the muscular fibres. Inflammatory processes, and others analogous thereto, commonly terminate in this way; thus arise the contractures consequent on traumatic myositis; those resulting from acute muscular rheumatism, the anatomical basis of which appears to be connective-tissue hypertrophy, the rheumatic wheal (rheumatische Schwiele); those consequent on syphilis, and probably also those arising from lead-poisoning; and lastly, those forms, the origin of which has been recently carefully studied by Mantegazza and myself, that commonly arise in muscles paralyzed by injury.[1] According as the influence of the nerves is, or is not, re-established, these contractures are merely transient, or become permanent. They may be particularly well observed in traumatic paralysis of the extremities and in severe facial paralysis. These paralyses have this common and characteristic feature, that, on electrical investigation, both the nerves and the muscles exhibit the peculiar reaction that I have termed "reaction of degeneration."[2] It is probable that this mode of origin of muscular contractures plays a certain though not very important part in the spinal paralysis of children, since in this disease some of the paralyzed muscles exhibit the reaction of degeneration.

In *neuropathic contractures* the persistent and frequently very marked rigidity and shortening of the muscles are caused by a stimulus affecting the motor nerves. The stimulus may affect

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[1] For the histological details the reader is referred to the section bearing on the subject in the chapter on "Paralyses."

[2] Entartungsreaction. Vide the chapter on Paralyses.

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various points of these nerves, and may be conducted to them from different parts.

1. *Peripheric stimuli* may affect the motor nerves directly, and thus occasion the contracture. This is seen in neuritis, in neuromata, as a result of the presence of foreign bodies, in gunshot injuries, and in similar traumatic lesions of the nerves. In such cases the contracture is strictly limited to the region of distribution of the irritated nerve fibres, and then other peripheric symptoms are almost always present, such as violent neuralgia, formication, anæsthesia, and not unfrequently paralysis.

2. *Peripheric sensory irritations* may excite the motor nerves by reflex action, and thus induce contracture (reflex contracture); to this category belong those contractures that are so frequent as a consequence of painful inflammations of the joints, articular neuralgiæ, and severe fractures.

3. Lastly, *centric irritations*, and especially cerebral diseases, are amongst the most frequent causes of neuropathic contractures; amongst these must first be mentioned the contractures that are so common in hemiplegic patients, and in regard to the origin of which various opinions are still held. They occur chiefly in those forms of hemiplegia that originate in apoplexy of the central ganglia and of the cerebral hemispheres; they affect only the paralyzed half of the body, and not only those muscles that are completely paralyzed, but also those which are in part capable of again recovering their functional activity. Certain groups of muscles are particularly disposed to be hemiplegically contracted, as the flexors in the upper extremity, and with increasing intensity towards the hand; hence the characteristic position of the arm in so many hemiplegic patients, in which the arm is drawn to the side, the forearm is flexed at a right angle, the hand is clenched and the fingers are only with the greatest difficulty capable of being extended. It is easy to show that the extensors are not entirely free from contracture, for if the arm be forcibly extended, and an attempt be then made to bend it, the same resistance is offered by the triceps as was previously presented by the biceps. In the lower extremities some of the flexors and some of the extensors appear to undergo contracture; the knee is extended and stiff, the foot is in a state of plantar

flexion, and the toes are strongly flexed. Hemiplegic contractures of this kind usually appear soon after the apoplectic attack; they are more or less strongly expressed in different instances, and sometimes they are completely absent; this is probably due to variations in the seat of the lesions, but as yet we cannot state with any precision what part of the brain must be damaged to lead to the production of contractures. The rigidity of the muscles usually disappears during sleep and gradually returns on awakening; it is almost always increased by voluntary movement, and may persist, even when the paralysis has considerably diminished, and when the antagonists of the contracted muscles are fairly capable of performing their functions.

The mode of origin of these hemiplegic contractures differs essentially from that of paralytic contractures, and the primary causes of the latter are rarely present in hemiplegic contractures, or aid in their production. Moreover, there can be no question of the occurrence of any myopathic alteration; the histological characters, the volume and the electrical excitability remain, as a rule, long unaltered in hemiplegic patients. The symptoms are apparently due to abnormal excitations, which affect the motor paths in the central organs. It was formerly generally thought that the contractures were produced in a direct manner, by irritation of the motor nerves consequent on the occurrence of secondary inflammatory phenomena, such as hyperæmia and sclerosis, in the immediate vicinity of the apoplectic clot. Hitzig, however, has lately endeavored to show, by a series of interesting observations, that hemiplegic contractures have a somewhat different mode of origin; he regards them as associated movements which represent only an abnormal increase of those associated movements that inevitably accompany all, even the simplest movements of the body. These associated movements are excited and co-ordinated in definite cerebral centres, distinct from the centres of voluntary excitation. If a condition of irritation exists in these parts, the voluntary excitations passing to them will lead to abnormally increased and abnormally distributed associated movements, and thus to contractures. The duration of these associated movements, which far surpasses that produced by the stimulus of the will, is explained by Hitzig as a faculty, gradually acquired by the morphological elements in the course of the disease, of detaining excitations conducted to or arising in them.

Whichsoever of these two opinions may be correct, we must in any case attribute hemiplegic contractures to abnormal central irritations. Contractures may therefore occur in all cerebral diseases, and particularly in localized affections, if they are capable of producing similar cerebral irritation: for example, encephalitis, abscesses and tumors of the brain, cerebral sclerosis, acute meningitis, acute hydrocephalus, etc. It depends on the seat and extent of these lesions whether the contracture is confined to particular muscles or groups of muscles, or whether it is more widely distributed, or, lastly, whether it appears in a hemiplegic form or not. We may here also include the contractures that frequently occur in chorea and other neuroses, and in hysteria, and also some of the congenital contractures which accompany congenital diseases and malformations of the brain. Further remarks upon this point will be found under the head of cerebral diseases.

Contractures also occur in *diseases of the spinal cord*, and these chiefly affect the lower extremities and the muscles of the trunk, and are usually symmetrical on the two sides of the body. They owe their origin in part to the direct irritation of the motor cells and fibres of the cord, but partly, and indeed most frequently, to reflex irritation. Here, also, the contractures are not unfrequently observed as associated movements in muscles which are wholly or almost wholly withdrawn from the influence of the will. The slightest degrees of spinal contractures are designated by the term "muscular tension." In this condition the muscles offer a certain resistance to passive movements, during which the tension is apt to be considerably increased by reflex irritation. In the higher degrees strong contractures occur, which are extremely difficult to overcome. They are most frequently observed in spinal meningitis, in myelitis, and occasionally in tabes. Contractures of spinal origin also occur in hysterical patients.

It is unnecessary here to enter more minutely into the *symp-*

tomatology of contractures, and we must refer for details to the manuals of general and orthopædic surgery, and to other chapters of this work. It is only requisite to mention the fact that the principal symptom is the deformity caused by the persistent approximation of the points of attachment of the contracted muscle, and that the tense projection and hardness of the contracted muscle, the limitation of the movements made by its antagonists, the inability to effect passive movements in certain directions, enable us, as a rule, to fix the position and the extent of the contracture.

No characteristic signs are obtained from the *reaction of the contracted muscles to electricity*, for this reaction depends upon the primary disease. (See the sections on Traumatic Paralyses, the Spinal Paralysis of Children, Hemiplegia, Myelitis, and Tabes.)

The extensibility of the muscles depends, in great measure, on the changes which the tissue itself has undergone. *Ceteris paribus*, it diminishes in proportion to the length of time that the contraction has lasted, to the state of development and degree of retraction of the connective tissue, and to the atrophy of the muscular fibres. It is well known that chloroform narcosis constitutes an excellent means for discovering these conditions, as well as for establishing the diagnosis of muscular contractures.

The *treatment* of contractures need not here be discussed at any great length, since it constitutes the chief object of orthopædic surgery, which endeavors to cure these affections by permanent extension (with the aid of bandages, compulsory positions, various apparatuses and machines), by tenotomy, by forced extension under chloroform, and by gymnastics. We may, perhaps, be permitted here to make a few remarks in regard to the *electrical treatment of contractures*, which not unfrequently proves of considerable value.

The electrical treatment of the primary paralysis is of the greatest importance in *paralytic* contractures; in proportion as the results of this are successful, the contractures will be removed. For further details the reader is referred to the sections treating of the several forms of disease.

Myopathic contractures, except in very recent cases, only seldom experience rapid improvement. Yet even in obstinate cases, as, for example, in torticollis of rheumatic origin, excellent results may be obtained by the energetic and protracted stabile action of the anode of a strong galvanic current upon the muscle, as well as by interruptions of the current and by the labile action of the cathode. The galvanic current also proves very effective in recent rheumatic forms, as does also the faradic when applied energetically to the skin. The myopathic contractures, occurring in consequence of traumatic and similar paralyses, yield most readily upon the removal of the cause of the paralysis; at a later period the nutrition of the muscles may be improved, and the contracture may then gradually be overcome by faradic, or, still better, by labile galvanic irritation.

Electricity is, however, most effective in the treatment of *neuropathic* contractures. It is least serviceable in contractures arising from direct peripheric irritation; in these cases everything depends on the possibility of removing that irritation. If the electric current be capable of accomplishing this, it will at the same time cure the contracture. Remak has obtained very favorable results with the galvanic current in the reflex contractures that occur so frequently as an accompaniment or as a sequela of articular inflammation (in which probably some inflammation has extended to the muscles). The best method of applying it consists in the transmission of a strong stabile current, which should then be occasionally interrupted, in order to produce muscular contractions, by which means the muscles are most readily caused to relax (attention being at the same time directed to the treatment of the diseased joint). Similar effects may also be obtained with faradic currents, partly by the action of strong local faradisation of the contracted muscles themselves, and partly by the faradisation of their antagonists (Duchenne).

Contractures of central origin are more difficult to treat, and the treatment may be either applied to the central organs or to the periphery, the former course being by far the more important; indeed, I have only seen permanent success result from treatment when it was directed to the brain and spinal cord, and when the central disease was benefited. It is obvious that only galvanic treatment directed to the centres can be employed; the methods are described under the several diseases. The contractures have been observed to diminish and gradually disappear in quite a number of cases of hemiplegia which were treated by galvanization of the head alone; and similarly the cure of cases of muscular tension and contracture by galvanization of the spinal cord, has not unfrequently been observed in patients suffering from tabes and myelitis.

Much useless trouble has been expended on the peripheric treatment of central contractures; the result is in general unsatisfactory, and such treatment requires much patience and perseverance. The best methods are the following:

(*a*) Faradisation or labile galvanization of the antagonistic muscles, assisted by passive extension of the contracted muscles, and appropriate extension apparatuses.

(*b*) Strong faradisation of the contracted muscles in order to effect their relaxation by over-stimulation. This may be advantageously combined with (*a*).

(*c*) Strong galvanization of the muscles with a descending stabile current, with subsequent frequent interruptions of the current, thus causing muscular contractions. According to Remak this is the best means of securing the relaxation of the contracted muscle.

My own experience has satisfied me that peripheric treatment is only to some extent useful when it effects improvement in the central disease. It is scarcely necessary to add that in many cases electrical treatment can only be regarded as an aid to other methods.

3. On Paralysis in General.

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By the term paralysis (akinesis) is understood the diminution or abolition of the faculty of exciting the normal function of the ACTIVE *organs of motion, that is, of the entire motor nervous apparatus and the muscles.*

By this definition the immobility occasioned by disease of the *passive* organs of movement, such as the bones, joints, and ligaments, is excluded from the conception of paralysis. On the other hand, however, the disturbances of movement arising from disease, loss of excitability, or atrophy of the muscles, the so-called myopathic paralyses, are included in the definition. It is not easy to discover why these should be excluded from the idea of paralysis, since the muscles form integral parts of the active motor apparatus, and represent the terminal apparatus of the motor nerves ; since, moreover, the symptom of motor paralysis can only be exhibited in the muscles ; and, lastly, since paralysis from disease of the muscular substance cannot be practically distinguished from that caused by disease of the motor terminal apparatus. It therefore appears justifiable to include in the general term paralysis those derangements of mobility which proceed from this part of the active apparatus of motion. Paralysis is obviously a mere symptom, like neuralgia, anæsthesia, and spasm, its characteristic being *defective or abolished muscular contraction whilst the power of making an effort of the will remains.* The determination of the genesis of paralysis does not necessarily belong to its definition, and it is not essential to its symptomatic conception whether the absence of the desired muscular contraction is occasioned by disturbance of the motor nerve apparatus (*neuropathic paralysis*) or by disease of the muscles themselves (*myopathic paralysis*). The conception of motor paralysis appears to us to be rendered clearer by this explanation. We shall of course be almost exclusively occupied here with the consideration of neuropathic paralyses.

But by our definition the immobility caused by deficiency of the central voluntary impulses (*i. e.*, by abulia) is excluded from the conception of paralysis. Ideas of movement and the will to carry them out and to transfer them to the motor apparatus, must exist for the conception of paralysis to be complete. Paralysis exists when the transfer of the impulses of the will (regarding the seat of which we are ignorant) to the most central motor apparatus (motor centres in the cerebral cortex of Fritsch and Hitzig?) or to the motor paths and the muscles, is hindered. On the other hand, the capability of producing an effort of the will in the mind may be extinguished (abulia), without the occurrence of paralysis; thus, for example, neither those affected with melancholia attonita, sleeping persons, nor patients under chloroform, can be regarded as paralyzed. We thus avoid the use of a term that is inaccurate, both in its psychological and psychiatric sense.

Paralysis can only appear in the form of a *diminution or abolition of the contractions of the various muscles of the body*, by whatever physiological means they may be called forth, whether by the influence of the will, by reflex, or by automatic excitation. The immediate result of the imperfect contraction is usually *defective movement or immobility* of certain normally movable parts of the body. It may here again be stated that every form of immobility is not attributable to paralysis, but that it may also originate in diseases of the joints, in anchylosis, contractures, and lack of will-power.

In order to distinguish simple diminution from complete abolition of muscular contraction, certain terms have been selected which distinguish the degree of motor disturbance present in any case. If the motility (voluntary movableness) be completely lost, so that no reaction follows a volitional impulse, *paralysis* is said to be present; if, on the contrary, only a more or less marked diminution of motility be present, so that weak and inadequate movements follow the volitional impulses, and fatigue sets in very promptly and readily, the term *paresis* is employed. In this case there are certain obstacles to motor excitation and conduction which cannot be entirely overcome by the influence of the will. Paresis may gradually pass into paralysis.

Pathogenesis and Etiology.—The first question that arises is, What part of the motor apparatus is affected by the changes leading to paralysis? And the reply is, simply, that paralysis may be induced by lesion of all parts of the active apparatus of movement, including the muscles. Everything that is capable of diminishing or abolishing the excitability and conductivity of the motor nervous apparatus, everything which can lower or abolish the excitability and contractility of the muscles, may lead to paralysis. The changes which may produce this effect are not in all cases accurately known. It is true that, in a great number of cases, well-marked and very important coarse anatomical lesions in ganglion cells, nerve fibres, and muscles have been demonstrated by pathological investigation, which sufficiently explain the abolition of excitability and conductivity; but there still remain a large number of cases in which no anatomical changes can be discovered, even upon the most careful examination, either in the nervous system or in the muscles, whilst experiments with certain poisons, which even in extremely minute doses rapidly produce well-marked symptoms of paralysis, render it highly probable that the negative result of our inquiries is not to be attributed entirely to the defects of our optical instruments. It may therefore be admitted that paralysis is occasionally the result of delicate molecular changes in the nerves and muscles, which escape detection with the means at present at our command. We cannot conceive how well-marked disturbance of function—which is obviously present in every case of paralysis—may occur, without the existence of a corresponding alteration of the molecular conditions, by which the nutrition of the tissue elements is effected. It is probable, however, that with the advance of our knowledge the number of the so-called functional paralyses will become more and more limited and confined to a relatively small proportion of cases.

In individual cases such coarse or fine lesions may affect very different parts of the motor apparatus, and thus lead to different modes of origin of paralysis. (We have here chiefly to speak of paralyses of voluntary striped muscles, but the general principles are equally applicable to automatic and reflex movements, the points relating to which will be included in parentheses). Observation and reflection teach that three great groups of paralyses may be distinguished.

1. *Paralysis may arise from destruction or functional incapacity of the motor central apparatus* (that is, the centres of automatic movements, or the ganglionic apparatus for reflex excitations). By the term motor central apparatus we mean those parts of the cerebrum in which volitional impulses are probably converted into motor excitations. It is probable that this central apparatus does not coincide with the centres for the will. Lesions of the centres for the will itself, which lead to abulia, are thus ex-

cluded, as well as those lesions which induce disturbances of consciousness and ideation, and consequently also of the will. Until quite recently we were almost completely ignorant of the seat of these proper motor centres, and even the epoch-making works of Fritsch and Hitzig, of Nothnagel and of Ferrier, have only in part dissipated the obscurity, leaving a number of questions open, which must be left to the future to solve. In regard to the pathogenesis of paralyses, the admission of the existence of these centres is in many respects rather a postulate required by theory than a well-established fact. It is probable that in addition to the now well-known motor parts of the cerebral cortex, parts of the ganglia at the base of the brain, as well as parts of the cerebellum, may be regarded as such centres. But for practical purposes it is sufficient to describe paralyses arising from diseases of these motor centres, which are already partly known, and are partly to be hereafter more exactly localized, as *central paralyses* in the strict sense of the word (in order to distinguish them from central paralyses of conduction, which will be immediately characterized). In such cases the motor paths of conduction, throughout their whole course, as well as the muscles, may remain completely intact.

2. Secondly, *paralyses may result from diminution or abolition of the conductivity of the motor nerves.* But inasmuch as these nerves are very long and widely distributed, and may at the same time be affected in very different ways, these forms of paralyses are by far the most frequent; they form the great and important group of the so-called *paralyses of conduction* (conduction-paralysis, Leitungslähmungen). They may arise from lesion of the motor fibres, which proceed from the motor centres in the cerebrum, and, passing through the peduncles and the pons, reach the antero-lateral columns of the spinal cord; they may also originate from some lesion in the paths for the conduction of motor impulses in the spinal cord, paths which run both in the so-called white columns and in the gray (kinesodic) substance; and, lastly, they may be due to some lesion in the motor fibres which leave the spinal cord by the anterior roots and run through plexuses and peripheric nerves to the muscles. Three kinds of paralyses of conduction are consequently distin-

guished, according to the particular segment of the motor nerves that is affected, namely, *peripheric*, *spinal*, and *cerebral paralyses of conduction.* (But inasmuch as the two latter are often included amongst "central paralyses," it is important that we should again warn against confounding them with "central paralyses," in the strict sense of the term. These are always cerebral paralyses, but it is not every cerebral paralysis which is a central paralysis, as there are also cerebral paralyses of conduction.)

3. Thirdly, and lastly, paralysis may result from *abolition of excitability and contractility of the muscles.* This may arise from poisons, degeneration, and other lesions. The stimulus proceeding from the motor paths is no longer capable of exciting the muscular fibres to contraction. This is the so-called *myopathic paralysis*, which, whilst it is often only a subordinate symptom, is frequently also of essential and most significant import as regards the course and termination of certain muscular diseases. This form will be discussed at greater length in the section on the diseases of the muscular system.

All the forms of paralysis are referable to one or other of these three modes of origin, and, from a pathogenetic point of view, three groups of paralysis may be advantageously distinguished: central paralyses, paralyses of conduction, and paralyses of the muscles. The usual division of paralyses, which differs from the one just given, has been chosen for practical reasons.

Amongst the true *causes of paralysis* which, from what has just been stated, may act on various segments of the active motor apparatus, *wounds* are at once the most common and the most intelligible. It is obvious that complete destruction or section of the motor nerves must occasion paralysis; and that the same effect must follow crushing, mechanical compression and similar injuries, by which the histological character of the nerves and muscles is evidently altered. The very severe effects of slight mechanical pressure are less intelligible, since it often leaves no perceptible traces in the nerves, no change of the electric excitability, and no secondary trophic disturbances; yet very obstinate paralyses often follow such pressure, as is seen,

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for example, after pressure applied to the musculo-spiral (radial, Henle) nerve, and it is impossible to deny that even a slight mechanical action may so change the molecular condition of the motor nerves as to abolish their power of conduction. The interesting experiments of Weir Mitchell[1] have furnished some information on this point; he found that a pressure of from 18 to 20 inches of mercury, exerted for fifteen seconds, was sufficient to completely interrupt the conduction of motor impulses (whether of the will or of electricity); after removal of the pressure, the conduction power soon returned, but the medullary substance of the nerve fibres at the part which had been subjected to pressure was entirely destroyed. It is clear that had the pressure been rather greater, or had it been allowed to act for a longer period, the axis-cylinder which was uninjured in this experiment, would have been seriously damaged, and would consequently have caused the paralysis to be still more persistent. There can be no doubt that paralyses from compression in man originate in this way. Experience teaches that motor nerves offer less resistance to such lesions than the sensory ones do; for, if mixed nerve trunks be exposed to an injury of this kind, the disturbance of motility is greater in degree and more obstinate than that of sensibility. It is unnecessary to mention all the possible forms of traumatisms that may lead to paralysis (over-extension, laceration, contusion, compression, gun-shot wounds, injuries from cuts, stabs, fractures, luxations, etc.). In discussing the several forms of paralysis we shall see that certain nerves are predisposed to particular kinds of paralysis from injury, and are thus comparatively frequently and typically attacked by traumatic paralysis. These *traumatic paralyses* are necessarily of most common occurrence in the peripheral nerves (the brain and spinal cord being protected by their bony investments from external injuries), and constitute a very well-marked type of this form of paralysis, the experimental study of which has greatly added to our knowledge of paralysis generally.

Next in frequency to traumatic paralyses are those caused by

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[1] Loc. cit., p. 111.

disease of the parts in the neighborhood of the nervous system, so far as these are capable of being produced by mechanical pressure, by compression, by over-extension, etc. In this way paralysis may be produced by exostoses, abnormal formation of callus, caries of the vertebræ, aneurisms, echinococci, enlarged glands, herniæ, all kinds of tumors, and many other conditions.

Diseases of the nervous system are amongst the most frequent causes of paralysis. They may act in different ways, either by directly destroying the nerve elements (ganglion cells and nerve fibres), causing them to undergo degeneration and atrophy, or by compressing them, and thus rendering them incapable of performing their function ; or, lastly, by modifying their condition, or so altering their molecular composition as to render them incapable of discharging their normal function. It is easily intelligible, consequently, in what manner this effect is brought about by inflammations, such as neuritis, myelitis, and encephalitis, by extravasations of blood, as in apoplexies of the brain and spinal cord, by local softenings, by scleroses, tumors of all kinds, gray and fatty degenerations, and atrophy, and by diseases of the membranes of the brain and spinal cord. In point of fact, paralyses, having very diverse seats, and varying greatly in degree, are amongst the commonest symptoms of all the above-named anatomical and pathological alterations.

Disturbances of the circulation in the various parts of the nervous system play an important part in the production of paralysis ; a sudden and complete arterial *ischæmia* acts most energetically on account of the trophic disturbances that result from total absence of blood, and that rapidly annihilate the excitability of the nervous apparatus. To this cause is due the hemiplegia observed in embolism and thrombosis of the cerebral vessels ; and to the same cause also is due the paralysis of the lower extremities in the well-known experiment of Stenson (compression of the aorta), which was first correctly referred by Schiffer to its true origin—ischæmia of the lumbar region of the spinal cord ; to the same cause, finally, is due the paralysis that occurs subsequently to ligature of the larger arterial trunks, when no branches are present by which a collateral circulation can be established. Such ischæmic paralyses supervene more slowly and with greater difficulty when due to causes affecting the peripheral nerves and muscles than in lesions of the central nerve-system. They are often incomplete, if the blood-supply be not entirely cut off, and they are often first observed when some great demand is made on the muscular powers. All the known causes of ischæmia may be followed by such ischæmic paralyses. *Venous stasis*, especially if complete, acts in a similar, though less intense manner; the trophic disturbance occasioned by the insufficient renewal of the blood (and perhaps also the direct toxic effects of carbonic acid and similar products of tissue metamorphoses) ultimately abolishes the excitability of the motor apparatus, and thus occasions paralysis. It does not appear to be certainly established whether *arterial hyperæmia* may also lead to paralysis. The transient phenomena of paralysis, usually stated to occur as one of the symptoms of congestion of the brain, are perhaps due to secondary œdema, or to the abnormal distribution of the blood in the different segments of the brain.

It is uncertain, also, how far alterations in the composition of the blood (anæmia, hydræmia, chlorosis, etc.,) may lead to paralysis, though it is by no means improbable, on *à priori* grounds, that great alterations in the quality of the blood may so modify the nutrition of the motor nerve apparatus as to occasion paralysis. It is through the blood, also, that *certain poisons* which are known to produce paralysis, act; these are probably conducted to the nervous system and to the muscles, and either produce acute trophic disturbances or chronic diseases of various segments of the active apparatus of movement, which render the performance of their functions impossible. Putting aside the narcotic and anæsthetic agents, such as opium, morphia, chloroform, alcohol, carbonic oxide, etc., which act chiefly on the organs of the consciousness and the will, it is chiefly the *vegetable alkaloids* and similar poisons, such as woorara, ergotine, nicotine, saponine, hydrocyanic acid, camphor, etc., which rapidly occasion intense and widely-spread phenomena of paralysis, while on the other hand certain *metallic preparations* only lead to paralysis after the lapse of some time, and often not till their toxic influence has been exerted for several years. The best known and most characteristic form of paralysis belonging to this group is that produced by lead, which is a not unfrequent result of saturnine poisoning. Similar paralyses are, however, seen, though much less frequently and in less typical form, in chronic poisoning with arsenic, mercury, and phosphorus.

Attempts have been made to refer *paralysis, occurring after acute diseases*, to certain changes in the blood, some of which, however, are, in part, hypothetical. Such attempts, however, are only applicable to a small proportion of those forms of paralysis that are now under consideration. It is, however, a fact that paralyses often occur coincidently with, or as a consequence of, a considerable number of acute diseases, and, particularly, after those that are broadly included under the term of infectious diseases, as the acute exanthemata, erysipelas, typhoid fever, intermittent fever, cholera, dysentery, acute articular rheumatism, and especially diphtheritis. The seat and distribution of these paralyses may vary very greatly. They sometimes present the form of hemiplegia, and sometimes that of paraplegia, or they may be limited to particular cerebral or spinal nerves, or even to particular muscles; they sometimes exhibit the characters of peripheral, sometimes those of spinal or cerebral paralysis (see below); and it is only the diphtheritic form of paralysis that presents any regularity in attacking different muscular regions, and in the course of the whole affection. From the observations that have hitherto been made, however, it appears that the paralyses occurring after acute diseases have, by no means, in all instances, the same pathogenesis, but that they are rather due to extremely different pathological conditions. It is probable that the alteration in the constitution of the blood, produced by the primary affection, and the consequent atrophic disturbances, are only very rarely, if ever, the cause of the paralysis. But we have sufficient grounds, from the general condition of the paralyzed parts, from their altered relations to electricity, and from the whole course of the affection, to permit us to hold that, in most instances, well-marked anatomical changes constitute the causes of the paralysis. It is true that such anatomical changes have only been demonstrated in a few instances.

Still, it is certain that various confirmatory facts have been obtained; thus Buhl found abundant cellular infiltration and hypertrophy of the nerve sheaths in almost all the roots of the spinal nerves, in a fatal case of diptheritis, and he attributed the paralysis to these conditions. Ebstein again found in one case of persistent weakness of the limbs, after typhoid fever, sclerosis of the central nervous system, and Westphal repeatedly found a disseminated myelitis of the gray and white substances, as a cause of the paraplegia occurring after small-pox. I have myself observed obstinate paralyses, which were clearly of spinal origin, after scarlet fever and severe puerperal disease. Very recently Bernhardt[1] has shown a circumscribed neuritis to be the cause of a paralysis of the radial nerve, arising in the course of typhoid fever. Further investigation will, in all probability, show that other lesions, such as extravasation of blood, inflammation, neuritis, tumors, arising in consequence of acute disease, may occasion paralyses.

The same is true in the case of paralyses occurring in *chronic infectious diseases, and in certain cachexiæ;* these paralyses, which may be referred partly, no doubt, to the defective quality of the blood, are chiefly caused by certain anatomical lesions of the nervous system. This is particularly the case in *syphilis*, which, most frequently of all these forms of disease, leads to paralysis. Syphilitic periostitis and exostoses, gummata, and chronic specific inflammations, diseases of the vascular system, etc., sufficiently explain the frequent occurrence of syphilitic paralyses, and their various seats. Besides these, which, for the most part, belong to the so-called tertiary period of syphilis, there are other paralyses that, according to Fournier,[2] belong to secondary syphilis, and chiefly affect the third, sixth, and seventh pair of cerebral nerves, or appear in the form of hemiplegia. *Scrophulosis* leads to paralysis most frequently by causing disease of the bones and periosteum, and less frequently in consequence of swelling of the glands. Paralyses, occurring in tuberculosis, are, generally, attributable to tubercular inflammation of the meninges, or to the development of tubercles in the substance of the brain and spinal cord. We are less able to explain the mode in which *gout* can cause paralysis, but it may be, or has been, referred to apoplexy, to gouty inflammation in the membranes and spinal cord, and to arthritic neuritis;—whether rightly or wrongly, must, for the present, remain an open question. A cause of paralysis which, notwithstanding its notorious frequency, has not as yet been sufficiently explained, is *catching cold;* this plays a great part in the etiology of all forms of paralysis, and its frequency has led to the establishment of a special group of paralyses, termed the "rheumatic paralyses," or better, perhaps, "paralyses from chill," a group which is insufficiently characterized, on the one hand, by the mode of its causation—by catching cold, and on the other hand by our ignorance of the anatomical changes lying at its root. Rheumatic paralysis may arise in all parts of the nerves, and in all nerve regions, but, perhaps, most frequently attacks the peripheric nerves, some of them, indeed, as the facial nerve, with a marked preference; and it is highly probable that the immediate occasion of the paralysis in such cases is a slight exudation or tumefaction in the neurilemma, or its slight inflammation, by which the nerve fibres are compressed. In accordance with the anatomical seat of these changes, and the accidental position of the nerves in more or less unyielding tissues, various severe and obstinate paralyses may arise. Central diseases, leading to paralysis, may also be induced by catching cold, as in the myelitis and meningitis spinalis, tabes, etc. These are, however, seldom included amongst rheumatic paralyses.

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[1] Ueber Radialislähmung. Archiv. für Psychiatrie and Nervenkrankheiten. Band IV., Heft 3, 1874.

[2] Leçons sur la syphilis, Paris, 1873, p. 805.

Those paralyses that arise from *exhaustion of the nervous system*, consequent upon inordinate exertion, have not as yet received any explanation. In this way immoderate muscular exertion, as, for example, severe forced marches, especially when performed under the influence of lively emotion (as during a battle), immoderate or unnatural satisfaction of the sexual appetite, continuous night watching with excessive mental exertion, and similar conditions, may induce paralysis. But this occurs indirectly by a more or less profound trophic disturbance of the nervous system (degeneration, atrophy, chronic inflammation, etc.) affecting the motor nerves, and gradually destroying their excitability and conductivity. What alteration lies at the root of the paralyses so frequently present *in hysteria* we do not know, and can only say that in the majority of cases hysterical paralyses are due to changes in the central nervous system, whilst it is still uncertain whether paralysis from peripheric changes occur in this disease. Moreover, the paralyses which sometimes appear in severe spasmodic diseases, *epilepsy*, *eclampsia*, *chorea*, depend for the most part on changes of the central organs, of the nature of which we are almost entirely ignorant.

We have, lastly, to mention a group of paralyses of very obscure etiology termed *reflex paralyses*, that is to say, paralyses which arise by reflex action, in consequence of some primary disease, injury, or irritation of the nerves at the periphery. Such paralyses—almost always occurring in the form of paraplegia, and more rarely limited to one extremity or one nerve trunk—have not unfrequently been observed after dysentery and other forms of intestinal disease, after vesical, renal, and uterine diseases, in consequence of gunshot wounds of the joints, and of injuries to the nerves, etc. These paralyses develop for the most part very gradually, and often only in the later stages, and after the long persistence of the primary disease; they occasionally disappear promptly after the removal of the cause, though usually they have attained such a degree of independence as to remain for a variable period thereafter. Attempts have been made to explain the origin of these paralyses on known physiological and pathological principles, but no generally accepted explanation has yet been advanced, nor will in all probability any be discovered, since there appear to be several modes of origin of reflex paralyses. Brown-Séquard believed he had discovered the nature of reflex paralysis in a reflex contraction of the vessels, caused by peripheric irritation, which produced ischæmia of the spinal cord, but this opinion has met with but little support. Jaccoud maintains that the paralysis is due to peripheric sensory irritation causing reflex exhaustion of the motor apparatus of the spinal cord. The experiments of Lewisson, who produced paraplegia in rabbits by violent contusion of the kidneys, urinary bladder, uterus, and certain parts of the intestines, which soon disappeared after the cessation of the stimulus, seemed to show that such transitory functional paralyses may result from powerful peripheral stimuli, though we are still ignorant of the precise mechanism of their occurrence. It is, however, probable that the cases belonging to this category form only a small proportion of the whole. Those cases are more intelligible in which inflammation creeping along the nerves from the periphery—neuritis ascendens—can be clinically or anatomically demonstrated, and which ultimately reaches the spinal cord, and there produces myelitis, the result of which is paraplegia.

Leyden, in particular, advances the opinion that this process plays the chief part in, and furnishes the origin of, those paraplegiæ which proceed from disease of the intestine, from disturbances in the urinary and sexual organs, and from peripheric injury of the nerves. Numerous and important facts, however, afford evidence that, quite independently of an ascending inflammation, or of a peripheric centre of irritation or of inflammation, circulatory and inflammatory trophic disturbances in the central segments of the nervous system may be produced by reflex action. Thus Tiesler found that after contusion of the sciatic nerve in a rabbit, a circumscribed myelitis appeared at its roots, whilst the intervening portion of nerve appeared to be entirely free from change. So also Feinberg, in a series of experiments on rabbits, saw inflammatory softening of the spinal cord arise at various periods after violent peripheric inflammation of the nerves (cauterization of the sciatic, refrigeration with ether spray) which caused death, preceded by symptoms of paraplegia, incontinence of urine, etc. In all cases the central portion of the sciatic remained free from any inflammatory alteration. Now, although all these experiments are neither sufficiently numerous nor sufficiently varied, they still furnish some evidence that inflammatory disturbances of the central nervous system may occur in a reflex manner—a view that is strongly supported by Benedict.

According to the above facts and opinions, we may state our present knowledge on the subject of reflex paralyses to be, that only a small proportion of them can be regarded as reflex functional disturbances of the motor apparatus in the sense of Lewisson's experiments, and that the majority depend on well-marked changes (disturbances of the circulation, and, above all, inflammation) of the central nerve apparatus. These alterations may be in direct and continuous connection with the peripheral lesion, owing to the presence of an ascending neuritis, or they may occur by reflex transference. Future and more exact observations must limit the regions in which these different forms of reflex paralysis occur. It need only be mentioned here that occasionally so-called reflex paralyses may proceed from thromboses of the pelvic organs conducted to the vertebral canal.

This short summary of the most frequent causes of paralysis must suffice to demonstrate in some measure the great variety and frequency of these forms of disease.

Pathology of Paralysis.

Symptoms.—The first and most remarkable symptom in the paralyzed parts is obviously the more or less complete *abolition of motility*, that is to say, loss of voluntary movement; the patients are incapable of voluntarily producing contractions in various muscles, of executing certain movements, and of assuming or maintaining certain positions. The paralyzed limbs hang motionless, they appear to the patient to be much heavier than before, and are either wholly (paralysis) or partially (paresis) incapable of being applied to their ordinary use. The general aspect of the disease is materially modified by the great variations that occur in the extent of the paralysis; thus a single muscle or group of muscles may be affected, or all the muscles supplied by a particular nerve or plexus of nerves may be rendered motionless; in other instances, all the muscles of one extremity may be paralyzed. Or, again, the paralysis may extend over one-half of the body, the arm, leg, and face of the same side being affected; this occurs chiefly in local lesions of the brain and on the side opposite to that of the cerebral disease, and is usually

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termed *hemiplegia* (though hemiplegia of spinal origin is also met with). Lastly, paralysis may affect both halves of the body symmetrically, and then generally commences in the lower extremities and spreads to the trunk and upper extremities. This is the commonest form of spinal paralysis, and is known as *paraplegia.* In rare instances, a high degree of paresis, or even true paralysis, affects almost all the muscles of the body.

The variety of the forms resulting from the differences that occur in the locality and extent of the paralysis is increased by the diversity of the *concomitant* and *secondary symptoms.*

If any trace of motility still remain, that is to say, if paresis only be present, the movements that can still be performed are not unfrequently accompanied by a remarkable trembling and uncertainty, and certain forms of ataxic motor disturbance may be readily referred to feebleness or entire loss of certain movements.

If complete paralysis be present, the condition of the paralyzed muscles varies in different instances; sometimes they are completely flaccid and relaxed, without the faintest trace of tone or contractility, and do not offer the slightest resistance to passive movements; in other cases, they are more or less tense and contracted (as, for example, in hemiplegic contractures, myelitic muscular spasm, etc.); in some instances, one condition succeeds the other, the primarily relaxed muscles gradually becoming tense and contracted. As we have already stated, in speaking of contractures (see page 379), these phenomena may be variously produced: sometimes they result from irritation which may develop more or less quickly in the neighborhood of the lesion causing the paralysis; sometimes they are due to simple trophic shortening of the muscles, the points of insertion of which are abnormally approximated; and sometimes they result from well-marked anatomical changes in the muscles, consisting chiefly of atrophy of the fibres and interstitial hypertrophy of the connective tissue.

The *relations and behavior of reflex movements* are of special interest. They vary considerably in different instances, as may be easily conceived, if we bear in mind how variously situated may be the different causes of the paralysis; the same is true in regard to *associated and automatic movements.* Complete absence of all reflex and automatic movements occurs only when there is interruption of the conduction in the peripheric motor nerves (or when the muscles are destroyed). It is very different, however, in most spinal and cerebral paralyses; in these affections reflex actions are preserved so long as the conductivity of the sensory fibres and their central connection with motor fibres capable of conduction are maintained, that is, when the cause of paralysis is situated on the central side of the reflex arcs, and these latter remain intact. In paralyses of the conducting fibres traversing the dorsal portion of the spinal cord, reflex actions liberated from the lower segments of the spinal cord may thus be preserved, and in cerebral paralyses reflex actions effected through the spinal cord and the medulla oblongata are undisturbed, but only so long as the reflex mechanism is not destroyed or rendered incapable of conduction by degenerative or other changes. In such cases, indeed, a considerable increase in the activity of the reflex actions may not unfrequently be observed consequent upon the removal of the inhibitory influence of the brain upon the reflex actions; and partly owing also to the establishment of conditions of irritation in the neighborhood of the lesion causing the paralysis. All these varieties may possess great diagnostic value.

The *associated movements* may also be preserved in the paralyzed parts in many cases where the paralysis is of centric origin, namely, in those where the cause of the paralysis is situated centrally to the centre of certain motor mechanisms, which, even under physiological conditions, are frequently accompanied by associated movements. Thus in cerebral paralyses, we not unfrequently see the paralyzed arm elevated when the patient yawns, coughs, or sneezes; and indeed the action is usually much greater than in health. If the view of Hitzig[1] be correct, such associated movements may also occur in paralyzed parts, when the impulses of the will traverse paths which lie below the cause of paralysis, and in which the physiological association and co-ordination of many muscular movements take place, provided these paths themselves are at the same time in an abnormal condition of excitability. The possibility of the occurrence of associated movements ceases the moment that the cause of the paralysis attacks those parts of the motor fibres where there are no longer communications with other motor nerves.

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[1] Auffassung einiger Anomalien der Muskelinnervation, I. Arch. f. Psychiatrie, III.

The same views must be held in regard to *automatic movements*, amongst which the movements of respiration are of the most importance in practice. The respiratory movements are rarely affected in peripheral paralyses, because the muscles subservient to this function are supplied by nerves having a widely different origin; here, at most, paralysis of the phrenics may be of considerable importance; of course, the movements of all those respiratory muscles which are included in the territory of a peripheral paralysis, are prevented; this, however, has but rarely a marked influence upon the movements of respiration as a whole. Where the cause of paralysis is spinal, disturbance of the respiratory movements occurs much more easily, especially if the lesion involves the lateral columns of the spinal cord in the dorsal and cervical regions, in which, as is well-known, the greater number of the respiratory nerves run. If the cause of paralysis affect the respiratory centre in the medulla oblongata, asphyxia is soon produced. Lastly, in paralyses of purely cerebral origin, respiration continues quite undisturbed, even with those muscles which are wholly withdrawn from the influence of the will. This depends immediately, and entirely, upon the normal function of the medulla oblongata.

Amongst the remaining automatic movements those of the iris must not be omitted, since they are disturbed in many forms of paralysis; thus in spinal paralyses affecting the upper segment of the cervical portion of the spinal cord, paralysis of the fibres of the iris, proceeding from the cilio-spinal centre, may occur, and thus lead to myosis; whilst, when cerebral paralyses extend to the region of origin of the third pair of nerves, mydriasis is one of the commonest symptoms. There is nothing, possessing any practical importance, to add in relation to the condition of the movements of the heart, the motility of the pharynx, œsophagus, etc.

On the other hand, the *discharge of urine*, and *the evacuation of the bowels*, deserve a short mention. These complicated processes, in producing which, reflex and voluntary movements co-operate, and the details of whose mechanism need not here detain us, may be remarkably impaired in various forms of paralysis. Retention, or incontinence of urine, confinement of the bowels, or their involuntary evacuation, may occur, and the presence of these symptoms may afford important aid in determining the seat of the disease to which the paralysis is due. For further details, we must refer the reader to diseases of the spinal cord, where, on account of the frequency of their occurrence, these conditions are most appropriately considered.

The condition of the antagonists of the paralyzed muscles fills a by no means unimportant position in the general picture of paralysis. Since they frequently pass into a condition of contracture, and their points of attachment are approximated in consequence of their elastic tension and the influence of the will, they occasion changes in the posture and position of the several parts of the body, in the expression of the face, etc., which are frequently very characteristic. These phenomena will, however, be better discussed when the several forms of paralysis are under consideration.

Disturbances in the sensory nerves occur in the most varied ways; this depends upon the accidental association of sensory and motor fibres in the same trunk, and upon the seat and extent of the cause of the paralysis. If the disease affects a peripheric mixed nerve trunk, the sensory paralysis or anæsthesia is, as a rule, equal in extent to the motor paralysis. There is a practical and important limitation to this rule in the fact that the motor nerves are usually more affected than the sensory ones, and that the re-establishment of sensation after its loss usually occurs much earlier than the recovery of motility, supposing the cause of paralysis to have been the same in both instances. It may indeed be not unfrequently observed in peripheric traumatic paralysis that, in spite of recovery of sensation, the motor paralysis remains irreparable. There is, of course, no sensory disturbance in peripheral paralysis affecting pure motor nerves.

Anæsthesia is frequently present in spinal paralysis, and depends upon the greater or less participation in the disease of the sensory parts of the spinal cord. There are, however, whole groups of spinal paralyses, as, for example, the paralyses of children and allied conditions, in which there are no disturbances of sensibility. The spinal origin of such affections may frequently be deduced from the intensity and extent, from the nature and kind of the sensory disturbance present; but for further details we must refer to the volume on diseases of the spinal cord. There is no strict rule in regard to the occurrence of anæsthesia in cerebral paralysis, because the sensory paths are more widely separated from one another in the brain than in the spinal cord; it therefore depends entirely upon the seat and extent of the cause of paralysis whether the sensory nerves are coincidently affected or not; both cases occur, but it may be observed here also that sensory disturbances usually disappear much more rapidly than motor ones.

It is clear that *symptoms of sensory irritation* occasionally occur, according to the seat and the nature of the cause of the paralysis, and we desire to recall to memory the violent neuralgic pains which occur in gunshot wounds of peripheral nerves, and the excentric pains which accompany many spinal and cerebral paralyses, sometimes with and sometimes without anæsthesia. Hyperæsthetic conditions are sometimes also present. All these variations may easily be explained by the irritation which is so often set up in the neighborhood of morbid processes causing paralysis, such as tumors, extravasations of blood, wounds, etc., and which partly produce pains and paræsthesiæ in paralyzed sensory nerves in accordance with the law of excentric projection, and partly extend to and implicate adjoining intact sensory nerves. In the same way the *paræsthesiæ* which are so common in paralysis (formication, numbness, creeping, burning, and allied sensations) make their appearance.

But few general statements having any practical value have been made in regard to the relations of *muscular sensibility* and the *sense of muscular effort.* There can be no doubt that disturbances occur in these sensations, and we shall give the points of most importance when discussing the several forms of paralysis. In true paralysis it is almost impossible to subject the so-called sense of muscular effort to any test, because the patient is unable to raise any weight, or to execute any voluntary movement.

The *intellectual faculties* are never impaired in peripheral paralyses, and it is only rarely that they undergo any considerable impairment in paralyses of spinal origin ; as a rule, they remain quite normal, providing no cerebral disease co-exists, or they only suffer secondarily at a late period ; if they then become affected it is probably due to the affection of the vaso-motor nerves, which, arising from the cervical portion of the spinal cord, or from the medulla oblongata, supply various vascular provinces of the brain. Disturbances of the intellectual faculties are, on the other hand, well-marked in those paralyses that result from cerebral lesion, though of course they will vary in degree and extent with the cause ; in such cases either impairment of memory and intelligence, loss of speech or difficulty of articulation, greater emotional excitability, strong inclination to laugh or cry without sufficient motive, great irritability and sensitiveness, or apathy, increasing to complete dementia, may be observed. Disturbances of the consciousness manifested by sopor, coma, delirium, etc., are sometimes seen at the commencement of acute cerebral paralyses, though they generally soon disappear again. The causal relation between these psychical disturbances and the real causes of paralysis is not always very clear; there is probably to some extent a direct extension of the disease to those segments of the brain which are subservient to psychical activity, and to some extent also a participation of these parts in the secondary diseases, inflammation, softening, sclerosis, which proceed from the primary focus of disease ; partly, too, a direct mechanical influence, occasioned by diminution in the capacity of the cranium; and lastly, there are disturbances of the circulation in certain segments of the brain, either from direct occlusion of arteries or veins, or from irritation or paralysis of vaso-motor nerves. For more exact information on all this, the reader must refer to the pathology of the brain.

The *vaso-motor and trophic disturbances* that occur so frequently in paralyses are of great importance. They also present great varieties, being sometimes very well marked and sometimes completely absent. We must refer for many points that belong to this subject to the account already given of trophic disturbances in neuralgia (see page 52 *et seq.*), and in anæsthesiæ (see page 218), and shall here only state briefly the most frequent disturbances of this kind that are met with, and endeavor to trace them to well-known physiological facts.

Physiology teaches us that in *peripheral paralyses*—that is to say, after section or compression of peripheral motor, and especially of mixed nerves—vaso motor and trophic disturbances are never absent. These manifest themselves in the form of hyperæmia, dilatation of the blood-vessels, and a local increase of temperature in the paralyzed part; which are all consequences of the coincident paralyses of the vaso-motor nerves. The rise of temperature passes generally, after a variable length of time, into a state of diminished temperature in consequence of the persistent atony of the vessels, which gradually leads to passive hyperæmia, with bluish red discoloration of the skin. In many cases, moreover, the paralysis interferes with the supply as well as with the return of the blood, since the accelerating influence of muscular contraction on the movement of the blood is abolished; hence follows the decrease of temperature observable some time after the occurrence of the paralysis, and the abnormal coldness with passive hyperæmia of the paralyzed parts and cyanosis. Associated with this, especially during the first few days, there is not unfrequently a widely-spread though slight œdema, and frequently also increased secretion of disagreeably-smelling perspiration. All these phenomena have been repeatedly observed in traumatic paralyses in man.

Whilst these are clearly vaso-motor disturbances, other remarkable changes are observed, which cannot be regarded as being simply due to paralysis of vaso-motor nerves, but belong rather to the category of trophic disturbances, and may be partly considered to be of an inflammatory nature. Amongst the most striking phenomena of this kind are those presented by the *skin*, which falls into a condition of diminished and imperfect nutrition, becoming flaccid and atrophic, and losing its vascular fulness; it often becomes as thin as paper, and, especially on the fingers and toes, smooth and bright (glossy fingers); not unfrequently vesicles appear, filled with a serous discolored fluid, which burst and leave behind them unhealthy, slowly-healing ulcers. The skin becomes less able to resist external injury, so that slight exposure to severe cold, the application of a mustard poultice, a wound, etc., occasion protracted ulcerations and nodular swellings. Bed-sores readily form and rapidly attain considerable dimensions. The characters presented by the epidermoidal structures of the skin are often very characteristic. The epidermis becomes hypertrophied, and separates in large scales, or it becomes thin, smooth, and glossy. According to Mitchell and Schiefferdecker, *the nails* undergo great alterations, becoming strongly curved and thickened, with deep longitudinal, or, more rarely, with transverse furrows, dark colored, and often partly detached from their matrix. The *hairs*, also, according to Mitchell, disappear from the skin of the paralyzed extremities ; whilst, on the other hand, Schiefferdecker constantly observed a greatly increased growth of hair. Amongst the more deeply-seated tissues, the changes occurring in the *muscles* deserve special attention ; they in all instances undergo a high degree of atrophy, the histological characters of which will be presently mentioned ; they may, indeed, almost entirely disappear, though they are still capable of almost complete restoration. In many instances the loss of muscular substance is apparently again made up by subsequent formation of adipose tissue. The bones do not escape the general failure of nutrition ; they become atrophied and *diminished in weight* (Reid, Mantegazza, Fischer) ; and, as a rule, when the paralysis occurs in youth, they cease to grow. In strong contrast with this is the hypertrophy and great increase in density or firmness of the connective tissue, which, on closer examination, shows itself as cirrhosis of the muscles and great increase of the interstitial tissue. The lymphatic glands enlarge and hypertrophy (Mantegazza), and according to Mitchell and Charcot, *the joints* become stiff, swollen, and painful.

The frequent gunshot wounds of the nerves received in the great wars during the last ten years show that in man also traumatic and similar paralyses constantly produce similar trophic disturbances in the tissues.

Phenomena of this kind are much less frequently observed in the paralyses that proceed from the spinal cord. In most of these cases vaso-motor disturbances are the most prominent, which is sufficiently accounted for by the course of the vaso-motor nerves in the cord. When completely divided, either by compression, laceration, or apoplexy of the cord, the symptoms of vascular paralysis are usually visible in the paraplegic parts; the paralyzed limbs possess a higher temperature (Hutchinson, Levier, Billroth, and others), which, however, after a time, usually falls below the normal, as in peripheral paralyses; this fall in temperature is accompanied by coldness and cyanosis of the affected part. This may, however, also be produced as a result of ischæmia consequent on irritation of vaso-motor nerves. Well-marked trophic disturbances of the skin, bones, and muscles are much less frequently observed in spinal paralyses, though even here they are of occasional occurrence; thus there is a group of spinal paralyses (to which the spinal paralysis of children and the allied forms of disease in adults belong), in which rapid and well-marked atrophy of the muscles, arrest of the growth of bone, and malformation of the joints, take place with great constancy. Moreover, severe bed-sores, with their distressing consequences, are amongst the most common symptoms of those spinal paralyses which are produced by disease of the greater part of the transverse section of the cord, and are accompanied by well-marked anæsthesia and vaso-motor disturbances; direct diseases of the bones and joints have also been regarded as dependent on spinal disease (Charcot). An increased growth of hair has occasionally been observed (Jelly). Most of these disturbances, however, disappear when the primary disease is cured.

The occurrence of vaso-motor and trophic disturbances is far more rare in *paralyses of cerebral origin*. Atrophy of the paralyzed muscles is scarcely ever observed; and even when the paralysis lasts for many years, they may remain quite unchanged in their size and anatomical characters. It is only in a few isolated forms of cerebral paralysis (as, for example, those having their origin in the pons, and similarly localized lesions) that distinct atrophy of the muscles has been noticed. As a rule, the growth of the bones is not in any way interfered with, and in those cerebral paralyses which occur in early childhood, the growth of the hemiplegic limbs is quite proportional to that of the opposite side. On the other hand, remarkable vaso-motor disturbances and considerable differences of temperature are not unfrequently observed in the paralyzed (usually hemiplegic) parts. In the early stages there is increased supply of blood and rise of temperature, which is easily accounted for by the fact that most of the vaso-motor nerves run in the pedunculus cerebri, which is also affected in many cases of paralysis (as in apoplexy), so that vaso-motor paralysis results. In the later stages atony of the vessels sets in, the circulation is retarded and still more opposed by the absence of active muscular contractions and the usual presence of contractures, so that passive hyperæmia, cyanosis, and a corresponding reduction of temperature are produced. With these vaso-motor disturbances, anomalies of the secretion of sweat, diminution of the supply of blood to the skin, doughy condition and attenuation of the skin, well-marked degeneration of the epidermis, are usually observed in cases of hemiplegia. Mitchell[1] reports that in recent hemiplegia the growth of the nails is at first completely arrested, then proceeds more slowly than on the sound side, and is again more rapid on the recovery of motility. The occurrence of acute and severe bed-sores has been occasionally observed in hemiplegia (Charcot).

The enumeration of the principal trophic disturbances occurring in paralyzed parts may be completed by a detailed though short description *of the histological changes undergone by paralyzed nerves and muscles* up to their complete recovery or complete irrecoverability. These conditions have, as we shall presently see, been made the object of much investigation in certain forms of paralysis, and this has furnished no slight aid for their clinical appreciation. The histological condition of the paralyzed nerves and muscles varies extremely in the different kinds of paralyses ; in a large series of cases, especially in most of those of cerebral origin (with the exception of paralyses having their origin in the pons), as well as in many spinal paralyses, probably also in all hysterical and in some forms of peripheric paralyses, no histological change can be for a long time discovered; the paralyzed nerves and muscles differ in no way from the healthy ones. It is only after the paralysis has existed for many years that we find here and there slight atrophy, the result of long inactivity, or there may be, especially where there is coincident contracture, moderate hypertrophy of the neurilemma, together with atrophy, brownish-red discoloration, and great softness of the muscles (Cornil).

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[1] American Journ. of Med. Sci. 1871.

In another series of cases, atrophy and attenuation of the nerve fibres, and especially of the muscles, may be observed, the latter becoming more slender and their primitive fasciculi smaller; the interstitial connective tissue also becomes somewhat hypertrophic, and not unfrequently the seat of a deposit of fat, which may to some extent mask the degree of the atrophy. This condition occurs especially in many forms of spinal paralysis.

Lastly, in a third series of cases remarkable histological changes occur in the nerves and muscles, and pursue parallel courses. These changes are typically presented in traumatic paralyses, and have there been most exactly studied by the aid of experiments; they occur in man in all traumatic and similar paralyses of peripheral nerves, in certain rheumatic paralyses or paralyses from chill, especially of the facial nerve, and also in all probability in certain spinal paralyses, especially in the already frequently mentioned spinal paralysis of children and the allied forms of disease.

The changes in the nerves and muscles must be separately considered.

The changes in the *nerves* have been long known, and have been most carefully investigated since the publication of Waller's suggestive works on physiology and anatomy. The following description is taken from the numerous memoirs of Waller, Schiff, Bruch, Lent, Hjelt, Philippeaux and Vulpian, Neumann, Erb, Hertz, Benecke, and many others who have occupied themselves with the processes of degeneration and regeneration consequent on traumatic lesions of the nerves.

Even during the first few days after any considerable injury of the nerves, such as section, compression, laceration, ligature, etc., coagulation of the medullary sheath takes place, and it gradually breaks up into longer and shorter cylindrical masses. These masses become progressively smaller, more rounded, and gradually converted into drops of various sizes, amongst which, at the end of the first week, a progressively increasing number of fine fat-granules may be observed. About this period the size of the degenerating fibres has undergone considerable increase, so that they appear broader than usual; the medullary masses and the drops of fat gradually unite and form larger and smaller irregular heaps, the smaller fat drops gradually preponderating and giving rise to the appearance of numerous fat-granules distributed through the nerves; after the third or fourth week there is a very considerable diminution of the medullary and fatty masses, which vanish in great part by absorption, but partly also by becoming converted into a homogeneous uniform mass, which fills the still remaining sheath of Schwann, and is traversed by the axis cylinder (Neumann). The degenerating fibres become gradually more slender, and ultimately there only remains a delicate pale band with irregularly undulating contours, still containing at certain points medullary and fat drops, and usually exhibiting a considerable increase in the number of nuclei. According to my own observations, this band is composed of the sheath of Schwann and the persistent axis cylinder, whilst Neumann considers it to be composed of the primitive sheath, filled with a homogeneous mass resulting from the conversion of the axis cylinder and of the broken-down medulla. Opinions are still divided in regard to the persistence of the axis cylinder, some admitting and some denying it; probably the case stands thus: in slight traumatic cases, such as simple compression, contusion, etc., in which the fibres have not undergone complete division, the axis cylinder remains until the power of conduction is re-established between the peripheral and the central portions of the nerve, by which it is then preserved from further disintegration. I at least have convinced myself that after simple contusion of the nerves in rabbits, as well as in paralysis of the facial nerve from compression in man, the axis cylinder is preserved for weeks in the peripheral segment of the nerve, and the remarkably prompt and early restoration of the motility which occurs in these experiments scarcely admits of any other explanation than that the axis cylinder persists. In serious injuries, on the other hand, where the nerves have been completely divided, and especially after excision of large portions, it is easy to conceive that the axis cylinder is completely destroyed in the later stages of the process of degeneration; even here, however, it still possesses a great power of resistence, may be demonstrated for a very long period, and is, according to Neumann, preserved in a modified form, which is a point of great importance in the regeneration of the nerve fibres.

Coincidently with these processes of degeneration in the nerve fibres, which spread with tolerable rapidity from the centre towards the periphery, *considerable changes also occur in the neurilemma.* Independently of the traumatic inflammation (indicated by swelling, abundant emigration of blood corpuscles, and hypertrophy, followed by cicatricial contraction of the connective tissue), which is always developed at the seat of the injury, there may be seen—associated with the progressive degeneration of the nerve fibres, and extending even to their finest branches—a remarkable increase and multiplication of the nuclei of the sheath of Schwann (Hertz), in the neurilemma itself, and an extraordinary accumulation of cellular elements, which, as we fully believe, may be for the most part regarded as emigrated white blood corpuscles. In the course of three or four weeks the greater number of these assume the character of fusiform cells, coincidently with which there is a progressive hypertrophy of the neurilemma; the external nerve sheath is greatly thickened, strong trabeculæ of connective tissue divide the several fasciculi of nerves from one another, and the hypertrophied connective tissue may even be seen to separate the individual fibres. As this process continues, the hypertrophied connective tissue becomes denser, firmer, and fibrous; true cirrhosis of the nerves is established, which is very clearly apparent when transverse sections are made. This hyperplastic condition of the neurilemma persists long after the regenerative process has begun in the nerve fibres, and it is probable that this latter is considerably prolonged and interfered with by this hypertrophy and retraction of the connective tissue.

The process of regeneration of the nerves, which sooner or later appears after traumatic and other lesions, is still imperfectly known, notwithstanding the numerous researches that have been made, and there is still a want of agreement amongst observers. It is, however, certain that in many cases regeneration occurs with great rapidity, in others much more slowly, but there in no uniformity of opinion in regard to the histological processes. The simplest case is that in which paralysis is caused by simple compression or contusion, but in which there has been no complete division of the nerve. We here see, as soon as the progressive absorption of the medullary and fatty masses permits sufficiently precise observations to be made, that pale bands in the old sheaths of Schwann extend from the healthy central ends of the nerve fibres into the degenerated peripheral portion; that these pale bands become gradually surrounded, from the central end outward, with a very narrow medullary sheath, which grows towards the periphery and becomes progressively broader. Hence, at a certain stage of the regenerative process, innumerable regenerated fibres may be met with above and a little below the point of injury, which are especially characterized by their slenderness, and the delicacy or narrowness of their double contour, and which are directly continuous below with the above-mentioned slender pale bands lying within the sheaths of Schwann. At this period the motility may already be partially restored. It is still doubtful in what way the restitution of the medullary sheaths and cylinder axes occurs at the point of lesion. Hertz is of opinion that the growing nuclei of the nerve sheaths in immediate proximity with one another, fill the hiatuses and re-establish the interrupted connection.

Very different is the process that takes place after complete division of the nerves, such as occurs in gunshot wounds, lacerations, etc., in human pathology, in which the two ends of the nerve are separated from one another by a longer or shorter interval, which must of course be in the first instance filled with granulation tissue. This gap has to be bridged over by nerve fibres of new formation, and the first thing in the process of regeneration is to establish connection between the centric and peripheral ends of the nerve fibres across this intermediate tissue. Great differences of opinion exist as to the mode in which this is effected. All observers agree, however, that the regeneration commences in the central segment, and that the newly-formed nerve fibres grow into the intermediate tissue ; and further, that a new formation of nerve fibres destined to join these occurs also in the peripheral segment, independently of the regeneration taking place in the central segment. But whilst some, as Hertz, Bruch, Hjelt, Oehl, and others, consider that the new nerve fibres originate from longitudinally arranged nucleated corpuscles (multiplied cells of the sheath of Schwann, or emigrated colorless corpuscles), both within the primitive sheaths and external to them; others, as Neumann, Eichhorst and Remak, consider them to arise from the fusion and regeneration of the homogeneous masses composed of altered medullary substance and cylinder axes contained in the sheath of Schwann, and exclude all idea of cells participating in the formation of new nerve fibres. A similar difference of opinion exists in regard to the mode of establishment of the connection between the central and peripheral fibres in the intermediate tissue between the two ends, Neumann believing that the central nerve fibres (of which, according to him, a whole bundle is formed in each of the old fibres) grow directly through the intermediate substance, and then join in some way with the peripheral fibres; whilst Hertz has satisfied himself that the serially arranged cells lying in the granulation tissue of the intermediate substance coalesce with one another longitudinally and become converted into nerve fibres, which join on the one hand with the central, and on the other hand with the peripheral fibres.

However this may be, it is certain, and of the greatest importance for the pathology of these affections, that regeneration of the nerve fibres does actually take place, that a reunion of divided fibres is effected by nervous tissue, and that thus the means for recovering from paralysis is afforded. Where the conditions for this regeneration are favorable, as in cases of incomplete division of the nerves, recovery takes place rapidly and completely ; if, on the contrary, they are less favorable, it takes place more slowly. The greater the distance between the divided nerve ends, the more they are turned from their normal direction, the broader and thicker the intermediate tissue between them, the smaller are the chances of a reunion and of a physiologically effective regeneration of the fibres. It is easy, therefore, to see that numerous cases occur in which the presence of such unfavorable external conditions render a reunion and regeneration simply impossible, and in which, consequently, the paralysis must remain incurable.

The interesting question, how it comes to pass that even after their complete division the physiological function of the nerves is completely re-established, and whether the divided extremities of each nerve fibre again unite, or whether the union is a matter of chance, has not as yet been subjected to anatomical investigation. We also know scarcely anything in regard to the conditions of the motor terminal plates in such processes of degeneration and regeneration.

In very close connection with the above-described alterations in the nerves, are a *series of changes that occur in the muscles* which have hitherto been but seldom the object of careful examination. Reid, in 1841, observed them closely, and noticed that remarkable atrophy took place in them. Valentine found attenuation of the fibres and indistinctness of the transverse striæ.

Mantegazza noticed in addition to these changes the growth of the nuclei of the muscle and of the intestitial connective tissue. I have myself carefully investigated and described the changes in the muscles occurring in traumatic paralyses, and my statements have been in great part corroborated by Vulpian, and very recently by Bizzozero and Golgi.

The principal and most striking change is incontestibly the *atrophy of the muscular fibres.* This first begins to be distinctly marked in the course of the second week, steadily advances during the following weeks, and may be easily recognized both in longitudinal and transverse sections, but especially in the latter. It is of course not quite equally pronounced in all the fibres, but fibres of very different breadths may be observed in paralyzed as well as in sound muscles; in the course of five or six weeks the average breadth of the fibres will be found to have diminished to less than half their normal size. When, however, regeneration of the nerves and restoration of the motility has commenced, the progress of the atrophy is arrested and the muscular fibres recover, though only slowly, their normal breadth. If the restoration of the nerves be impossible, and the paralysis consequently be incurable, the atrophy progresses; the several fibres gradually disappear, and there ultimately remains only the hypertrophied connective tissue. This termination, however—the complete disappearance of the muscular tissue—only takes place after the lapse of many months, and frequently only after several years.

In addition to the atrophy, several other changes may be observed in the fibres. The *transverse striæ become less distinct,* less sharply defined, they are more closely approximated and more delicate, though they do not entirely disappear. The fibres frequently appear slightly cloudy, though they never in the earlier stages present distinct granular or fatty degeneration. At this period the muscle-nuclei exhibit notable multiplication, which commences in the course of the second week, and soon after advances to such a height that where previously a single nucleus might be found there are now collections of from six to eight, which, especially in the atrophied muscles, are crowded together, and are much more readily visible without the employment of re-agents than in healthy muscles. Lastly, *a chemical change may be demonstrated in the contractile substance,* which is rendered evident by a tendency on the part of the fibres, increasing with the duration of the paralysis, to undergo, after injury or death, the peculiar change known under the name of "waxy degeneration"—a change which in the earlier stages of the paralysis is only a post-mortem phenomenon, but which at a later period perhaps takes place during life, and accelerates the disintegration and resorption of the fibres.[1]

The changes in the interstitial connective tissue, which are, also never lacking in the muscles, commence at about the beginning of the second week, with a moderate accumulation of cell-elements (emigrated white corpuscles?), so that, in many places, the appearance is presented of granulation-tissue having been formed between the muscles. This is particularly well-marked around the blood-vessels and the degenerated nerve trunks, and is everywhere easily discoverable for several weeks. From the sixth week onwards, the cells gradually disappear, after first becoming, for the most part, fusiform, and changing into the dense, wavy connective tissue, which is rapidly forming. This *hypertrophy of the interstitial connective tissue* can also be recognized at as early a period as the second week, from which time it steadily increases week by week, new material gradually assuming the form of dense, wavy bands of connective tissue of considerable breadth, which penetrate the muscles and separate the several fibres from each other by strong septa. The muscles are thereby rendered denser and firmer, and the great increase of the interstitial tissue may be particularly well seen in them on transverse section. The newly-formed tissue subsequently undergoes cicatricial retraction, and there can be no doubt that the contractures that occur so frequently after such paralyses, are chiefly caused by this condition, and hence are myopathic.

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[1] Compare *Erb*, Bemerkungen über die sogenannte wachsartige Degeneration quergestreifter Muskelfasern, Virchow's Archiv., Bd. 43, 1868.

The various changes that have now been mentioned, generally pursue a retrograde course, when complete regeneration of the nerves takes place. This, however, is a slow and gradual process, and indeed, slower in proportion to the degree to which the changes had previously advanced, and the more protracted the duration of the paralysis; and it is easy to understand that the great hypertrophy of the connective tissue, constituting a true cirrhosis of the muscles, proves a serious impediment to the rapid recovery of their normal size and activity, and sufficiently explains the tedious process of the cure of such paralyses in man.

In incurable cases, on the other hand, the hypertrophied connective tissue becomes progressively more and more dense with the advance of the atrophy and the final disappearance of the muscular fibres; the result of which is, that in the course of some months, or years, the muscles are entirely converted into flattened cords of connective tissue. In some instances this tissue is the seat of the deposit of fat—fat-cells appear more or less abundantly in it; and, finally, in place of the atrophied muscles, true adipose tissue may be found.

The changes that have been above described are visible, even to the naked eye, in the paralyzed muscles. During the first few days, in consequence of the large blood supply they receive, they are red and swollen, and of somewhat firmer consistence than natural. They become paler and paler as the atrophy of the fibres and the hyperplasia of the connective tissue increase, and at the same time they increase in firmness and toughness, whilst their color passes from grayish-red into the tint of dense connective tissue, which again, owing to the abundant infiltration of fat, may become converted into the pale yellow of adipose tissue. With this last change the consistence of the tissue usually undergoes diminution.

As has already been stated, all of the changes just described in nerves and muscles occur in the best marked and most typical form in paralyses of traumatic origin, whether produced experimentally or resulting from accident (such as gun-shot injuries, cuts, contusions, etc.). There is no doubt that these changes—the existence of which, as we shall immediately proceed to show, can be clinically demonstrated by electrical tests —may also be observed in all those forms of peripheral paralysis that are analogous to traumatic paralysis in being accompanied by a want of the anatomical integrity and conductivity of the nerves. Amongst these may be included the paralyses caused by energetic compression of peripheral nerves by tumors, extravasations of blood, formation of callus, hyperplasia of connective tissue, rheumatic exudation, etc. ; and the occurrence of the changes which we are now engaged in considering invariably points to some serious peripheral lesion.

Recent researches have rendered it probable that also in certain forms of central paralysis, identical, or at least very similar, changes, occur in both the nerves and the muscles. Thus it is believed that in the so-called spinal paralysis of children, and allied forms of disease, in which such alterations occur with tolerable regularity, the lesion is located in the anterior half of the spinal cord, and that certain ganglion cells in the anterior horns stand in such relations to the trophic processes in nerves and muscles, that, when they (the cells) are paralyzed or diseased, the changes in question may readily follow as a result. For further consideration of these facts, which are so important from a diagnostic point of view, we must refer to the section on diseases of the spinal cord and trophic nerves.

Lastly, we must not omit to mention that very similar histological changes have been observed in lead paralysis,[1] though the seat of this paralysis is still obscure; and, further, that the changes in the muscles that have here been described possess an extraordinary similarity to those observed in progressive muscular atrophy, so that from an histological point of view they may be regarded as identical.[2] Nevertheless, a considerable clinical difference exists between them in the circumstance that in the latter disease the motor fibres are preserved intact until a very late stage of the muscular affection. These facts, into the details of which we cannot here enter, could not, however, be omitted, because they serve materially to modify the differential diagnostic value of the clinical evidence of the anatomical changes with which we have just been occupied.

If the question of the *close connection of the above-described vaso-motor and trophic disturbances with the paralysis*, or rather with the cause of the paralysis, must be briefly referred to, it must only be to mention the numerous difficulties and obscurities which still surround this important point. Very little positive knowledge has as yet been obtained, and different opinions are held on nearly every point, the most contradictory opinions being on the most important points. Nothing has been irrefragably established, and the facts that have been obtained are for the most part capable of several interpretations, whilst experimental researches have to contend with what at present appear to be insurmountable difficulties. In any case, numerous experiments must still be made to enable us to give a satisfactory explanation of the trophic influence of the nervous system.

The vaso-motor and associated disturbances occurring in paralysis are most easily explained; they result simply from conditions of paralysis or irritation of vaso-motor nerves, which may exist or be absent, according to the seat, the kind, and the extent of the cause of the paralysis. Irritation of the vaso-motor nerves, and ischæmia, consequent upon this, are, from the nature of the case, quite rare in paralyses; paralysis of the vaso-motor nerves is, on the other hand, of more frequent

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[1] *Gombault*, Archives de physiologie normale et pathologique, 1873, Sept.

[2] Friedreich, Ueber progressive Muskelatrophie, Berlin, 1873, p. 173.

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occurrence, and in the first instance leads to hyperæmia, with slight œdema, and subsequently to general atony of the vessels, cyanosis, and defective nutrition. These symptoms may be chiefly expected in those cases where the vaso-motor nerves are coincidently affected by the primary cause of the paralysis, and they are almost certain to occur in cases of paralysis of peripheral nerves, where the vaso-motor nerves run in the same trunk. They are less frequent in paralyses originating in the spinal cord, because the two sets of fibres are then probably more or less widely separated. Lastly, they are very seldom seen in cases of cerebral paralysis, in which the vaso-motor nerves often remain quite intact, since in the brain the vaso-motor run more or less completely detached from the motor nerves. This is in complete accordance with clinical experience.

It is otherwise, however, with true trophic disturbances, such as nerve degeneration and disintegration, multiplication of nuclei, atrophy, and degeneration in muscular fibres, plastic infiltration and hypertrophy of connective tissue, inflammatory affections of the skin and joints, modified growth of hair and nails, atrophy of bone, and bed-sores. The general pathological significance of all these processes is indeed difficult to determine; many are no doubt to be regarded as a kind of inflammatory disturbance, like the chronic inflammation which in other organs leads to cirrhosis, whilst others must be considered examples of simple atrophy or of atrophy with degeneration, or of abnormal hypertrophy. Still more difficult is the explanation of the connection of these disturbances with the primary lesion. Even in what appears to be the simplest case—peripheric traumatic paralysis—the relations are so complicated that the most various explanations may be advanced and maintained.

The view that might appear most probable is, that a traumatic inflammation is propagated from the place of injury and extends along the peripheral nerves to the muscles, and occasionally to other tissues; and this view has recently been cleverly maintained by Friedreich.[1] We are, however, ourselves opposed to this explanation on various grounds: First, on account of the exclusively centrifugal extension of the process; secondly, on account of the almost coincident occurrence of the changes in the muscles lying in the immediate proximity, and in those that are most remote from the point of lesion; lastly, on account of the well-known fact, which has been frequently made use of in physiological investigations, that after section of particular roots the nervous degeneration extends through the plexus and towards the periphery, remaining limited to the original fibres, which is scarcely conceivable on the supposition of an inflammatory process taking place in the connective tissue of the nerve sheaths.

According to the facts and observations that have thus far been accumulated, we must consider the most probable explanation to be that the *separation of the peripheral parts from the central nervous system is the essential cause of all the disturbances.* How this takes place is in many respects still obscure. Vaso-motor influences in any case probably play a great part in their production; they explain the hyperæmia, the swelling, and towards the close also the emigration of colorless blood corpuscles, but they do not explain all; they do not explain the degeneration of the medullary substance of the nerve, nor the atrophy and multiplication of the nuclei of the muscular fibres. We are compelled to admit also the probable existence of direct trophic influences proceeding from the nervous system. Whether these are transmitted through the motor fibres, or through special nerve fibres (trophic nerves), must be left undecided. We are still almost entirely ignorant of the precise mode of action of the normal or pathological influences exerted through these nerves, and must defer a more complete discussion to the section on trophic neuroses.

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[1] Progressive Muskelatrophie, etc., Berlin, 1873.

If this view is correct, it follows that there are certain trophic centres in the central nervous system (probably in the spinal cord, but in part, also, in the brain), the separation of which from the peripheric parts occasions in the latter trophic disturbances in the widest sense of the term. It is, however, self-evident that similar trophic disturbances must also arise when these trophic centres become diseased, or are in any way injured. And we may thus explain the similar trophic disturbances that occur in the spinal paralysis of children and allied diseases. These are, however, usually limited to the motor apparatus (nerves and muscles).

Charcot, on the other hand, is inclined to assign an important part to irritation in the production of such trophic disturbances, and has attempted to trace back the greater number of trophic disturbances, both in peripheral and centric nerve lesions, to abnormal conditions of irritation; but the reasons he gives for this opinion, especially in relation to peripheral traumatic paralyses, appears to us to be exceedingly unsatisfactory.

It thus appears that our knowledge is extremely uncertain in regard to the whole of this subject. No explanation hitherto given has been generally satisfactory, nor is any applicable to all cases. Several circumstances, perhaps, co-operate in producing the particular effects observed, as the direct propagation of irritation, the removal of centric trophic influences, vaso-motor influences, and irritation of the nerves. Perhaps, also, a certain rôle is played by reflex processes, quite independently of the possibility that different trophic disturbances may be caused *indirectly*, when paralysis and anæsthesia are present. It is only by long and critical observation, and, above all, by experiments, that the question can gradually be solved. A short provisional communication of Nothnagel's[1] must not be undervalued, for he observed that a slight injury of a certain part of the surface of the brain of the rabbit was succeeded by a regular attack of meningitis, usually bi-lateral, very seldom upon the punctured side alone, and occasionally only on the opposite side.

Electrical examination of the paralyzed nerves and muscles has lately become of great importance in elucidating the pathology of paralyses, and in part also in determining their differential and local diagnosis. In different forms of paralysis very characteristic alterations of electrical excitability are observed, and in certain forms this mode of examination enables us to recognize and follow with precision the histological changes taking place, so that it is now quite indispensable in establishing the exact diagnosis of paralysis. Indeed, the diagnostic value of electrical investigation cannot be overestimated ; at the same time, he who believes that for the purposes of an exact diagnosis of a paralysis a single examination, however careful, is sufficient, will frequently be liable to error, since electrical investigation in many cases affords no very valuable information, and it is only in certain forms of paralysis that its results are free from ambiguity and permit perfectly definite inferences to be drawn ; whilst in all cases positive conclusions can only be arrived at by comparing the condition of electrical excitability with other clinical symptoms. The value of electrical investigation is so great that we cannot forbear to enter into considerable details in regard to it, though we must refer for particulars of the mode of research and of the physiological relations to the remarks that have been made in the introduction (page 271). We shall therefore here only briefly notice the results that may be obtained, and their relations to the several forms and kinds of paralysis, and to certain histological changes.

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[1] Centralblatt für die Med. Wiss., 1874, No. 14.

The results may be arranged in several groups.

In the first place, in many paralyses *scarcely any alteration of electrical excitability* is observed ; the nerves and muscles react normally both to the faradic and to the galvanic current. This is the rule in many paralyses of cerebral origin, as, for example, in hemiplegiæ after apoplexy or cerebral embolism, in those due to cerebral tumors, etc.; in many spinal paralyses, as, for example, in chronic myelitis ; and, lastly, also in many peripheral paralyses, both in those of rheumatic origin, as in slight facial paralysis, and in slight traumatic paralyses, as in paralysis of the radial nerve from pressure.

The second group includes those in which there are *simple quantitative changes of the electrical excitability.* In these the mode of reaction, law of contraction, etc., are normal, but the intensity and strength of these acts are altered, and are either diminished or increased.

(a) *Simple increase of electrical excitability* manifests itself on the application of faradic tests by the nerves and muscles reacting more energetically to the current, and by an increase of the distance of the secondary coil at which minimum contractions are produced, or by increase in the amount of contraction with the same strength of current; or, if the galvanic current be employed, by the occurrence of cathodal closing contractions (KaSZ) with very feeble currents; by the conversion of a simple contraction into a cathodal closing tetanus (KaSTe), when the strength of the current is slightly augmented; by the early appearance of the anodal opening contraction (AnOZ), and by the ready establishment of cathodal opening contraction (KaOZ); and, lastly, in rare cases, by the occurrence of anodal opening tetanus (AnOTe). As an additional characteristic, the increase of the so-called secondary excitability (Brenner) may be mentioned, and lastly the frequent disproportion between the motor and the sensory reaction, which is not unfrequently observed in such researches.

These changes, if they be not very intense in degree, are usually only to be ascertained with certainty and facility in cases where a comparison can be made with the symmetrical nerves and muscles of the opposite and healthy side, in other words, in cases of hemiplegia. In bilateral paralyses, paraplegiæ, etc., greater difficulty is experienced, and a comparison with healthy persons can only be instituted with very special precautions, which I have elsewhere given in detail.[1]

Simple increase of electrical excitability is of rare occurrence in paralysis; it occurs to a moderate extent in certain cerebral paralyses, and especially in the early stages (hemiplegiæ from various causes); it occurs also in many forms of spinal paralysis, especially in certain cases of tabes, though more rarely than is generally admitted. Lastly, it also occurs, rarely and quite transiently, in certain peripheral paralyses, as in the first few days of rheumatic facial paralysis.

(b) *Simple diminution of electric excitability* is manifested on faradic investigation by the diminution of the distance of the secondary coil at which minimum contractions are produced, and may, in extreme cases, proceed to complete extinction of faradic excitability. In investigations with galvanic currents, cathodal closing tetanus (KaSTe) first disappears, and cannot be induced by any ordinary strength of current; anodal closing contraction (AnSZ), and anodal opening contraction (AnOZ), then disappear; and, finally, cathodal closing contraction (KaSZ) can only be obtained with the strongest currents. Complete loss of galvanic excitability may ultimately occur.

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[1] Archiv für Psychiatrie und Nervenkrankheiten, Bd. IV.

This alteration is more common in paralyses than the preceding one; in cerebral paralyses it is, however, somewhat rare, especially in those which proceed from lesions of the crus cerebri, as, for example, in bulbar paralysis; it occurs, however, —and this is important as distinguishing bulbar paralyses from peripheral paralyses—not at an early period, but always in the later stages of the disease, and proceeds without any qualitative changes in the excitability. Moreover, this change is observed in certain spinal paralyses, particularly in those which are accompanied with simple atrophy of the muscles, and it is indeed the physiological expression of that atrophy; as, for example, in certain forms of myelitis, and in many spinal paralyses after acute diseases. The diminution and even complete abolition of electrical excitability occurs also in certain peripheral paralyses as the symptom of a large series of qualitative changes of the electrical excitability, and, lastly, as the terminal symptom of a complete cycle of changes in incurable cases, as we shall immediately proceed to show. Lastly, it accompanies the gradual wasting of the fibres in progressive muscular atrophy, and stands in direct relation to the area of the transverse section of the contractile substance still preserved in the atrophied muscles; with the complete disappearance of the fibres the electrical excitability also entirely vanishes.

The third group, lastly, includes the *qualitative and quantitative alterations of electrical excitability* which have recently become the object of much clinical and experimental study, and for which I have suggested the term "reaction of degeneration"

(Entartungsreaction).[1] The uncommonly characteristic course of this affection and the constancy of its occurrence in certain forms of paralysis confer upon it a high degree of diagnostic and prognostic value. After Hallé[2] had made an observation on this point, in Paris, towards the close of the last century—an observation, however, which was subsequently forgotten—and after Remak, in his "Galvanotherapie," had incidentally reported some similar cases (1858), Baierlacher[3] first published the history of a case of facial paralysis in which the muscles appeared to be completely inexcitable by faradic currents, whilst they reacted with unusual readiness to the galvanic current. This observation excited great attention, and numerous similar cases were soon published by Schulz, in Vienna, M. Meyer, Grünewaldt, Brenner, Neumann, Ziemssen, Eulenburg, Erdmann, Bärwinkel, Runge, myself, and many others; from all which it appeared that these changes in excitability were not limited to facial paralyses, but occurred in paralyses of all the various nerves. I was the first to undertake an experimental investigation of the subject, which has led to numerous important results. Coincidentally with my work appeared the treatise of Ziemssen and Weiss on the same subject, which confirmed my statements on all essential points. Since then numerous clinical observations, amongst the most important of which are those of Brenner,[4] as well as various isolated experimental researches have been made, which now enable us to give a tolerably complete exposition of this highly interesting question.

The first essential point resulting from these investigations is the fact that *nerve and muscle must be sharply distinguished from each other*, because the course of the alterations in the excitability in the two is wholly different. Want of attention to this fact has been the occasion of numerous errors and much misapprehension, which the employment of some precaution and care in conducting the research enables us now easily to avoid. The course of the alterations here referred to in the excitability is as follows.

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[1] Volkmann's Sammlung klin. Vorträge, No. 46.

[2] Onimus et Legros. Traité d'Electricité méd., p. 571.

[3] Bayr. ärztlich. Intelligenzblatt. 1859.

[4] Unters. u. Beobacht. auf dem Gebiete d. Electroth., Bd. II., 1869.

In the *nerve*, a short time after the attack of the paralysis (beginning with the second or third day, and preceded in rare cases by a slight increase of electrical excitability), a continuous and equable diminution of both the faradic and the galvanic excitability is observable, without any obvious qualitative change. This diminution is manifested, on the one hand, by the fact of a stronger current being required to produce the minimum contraction, and on the other hand by a considerable diminution of the maximum contraction obtained with the strongest current. At the end of the first or in the course of the second week (from the seventh to the twelfth day), *the excitability wholly disappears*, so that electric currents of ordinary strength, whether faradic or galvanic, when applied to the nerve, no longer occasion a trace of contraction. The diminution begins in that portion of the nerve lying nearest to the lesion, and extends very rapidly to the periphery. (There is, of course, total inexcitability by electric stimulation *above* the paralyzing lesion, from the very commencement of the paralysis.) The extinction of the electrical excitability may be lost for a variable period, and in incurable cases is, of course, permanent. Its restoration commences as soon the process of regeneration of the nerves has advanced to a certain point, and the first traces of both faradic and galvanic excitability are nearly coincident in point of time; it appears first in the central segments of the nerve, and spreads slowly to the periphery; it augments equably, but very gradually, and always without qualitative changes, in response to both kinds of currents, remaining for a very long time below the normal standard; when the cure, as regards voluntary movements, appears to be already quite complete, a distinct diminution is usually still perceptible in the faradic and galvanic excitability of the nerves.

During the early stages of the commencing regeneration, the interesting and apparently paradoxical symptom is presented of voluntary movements being effected through the paralyzed nerves at a time when they are still completely incapable of reacting to the electrical stimulus; that is to say, there is *restoration* *of motility with persistent loss of electrical excitability.* In other words, the nerve is for a certain time capable of conducting impulses of the will proceeding from the centre, whilst it is still incapable of responding to electrical stimuli. The duration of this stage varies, lasting in some cases only a few days, but in others for many weeks, the difference depending on the rapidity of the regeneration and the distance of the part of the nerve tested by electricity from the seat of lesion. This fact has been often falsely interpreted on account of insufficient attention being bestowed on the difference between the excitability and the conductivity of the nerve. I have discovered an explanation of this remarkable circumstance experimentally, in the fact that, at a time when the reunion of the central segment of the nerve has been effected with the peripheral, and when consequently young nerve fibres have grown through the point of lesion, though no distinct regeneration of the medullary sheath has taken place, that at this time the regenerated fibres are capable of conducting central excitations, though they are still inexcitable by electricity. The young, slender, regenerated nerve fibres above described are thus at a certain stage of their regeneration (which is histologically characterized by the complete absence or very small development of the medullary sheath) quite capable of conduction, but are not electrically excitable. Experiment proves that this capability of conduction is not limited exclusively to excitations liberated by the will, but may occur with any kind of irritation, for if the electrical, mechanical, or thermic stimulus be applied to the nerve above the point of injury, reaction takes place, because the irritation thus produced is conducted through the peripheral regenerated portion, although this is itself inexcitable by irritation applied below the point of injury. This explains why the stimulus of the will, which acts above the point of injury, can liberate muscular contractions at a time when the peripheric segment of nerve is still inexcitable. The difference between the activity of the stimulus of the will and that of electricity is thus referable simply to a difference in the place and point of application of the stimulus. All stimuli affecting the nerve above the point of injury are capable of acting at a time when the nerve segment below the point of injury is still unexcitable by the electric current. I have frequently been able to confirm this in traumatic paralyses occurring in man.

Not the smallest doubt can be entertained that the proof of the distinct and separate nature of conductivity and receptivity in the peripheral nerves, afforded by the foregoing fact, gives a clue to the explanation of the phenomena with the consideration of which we have just been occupied. This proof possesses also no small physiological interest, since it decides a question that Schiff[1] has discussed on several occasions and experimentally attacked without finally solving it, and which, as it would appear, has been finally solved physiologically by an interesting experiment of Grünhagen;[2]—namely, the question of the difference between the receptivity and conductivity of peripheral nerves.

Much more complicated phenomena are presented by the muscles, since they are not, like the nerves, affected equally by faradic and galvanic currents, but exhibit quite different phenomena when excited by the two kinds of electricity.

Paralyzed muscles behave in an almost exactly similar manner to degenerated nerves, when exposed to the *action of faradic currents*, that is to say, a *progressive diminution of excitability* occurs towards the end of the first week, which usually rises to *complete extinction* in the course of the second week, so that the strongest faradic currents occasion no trace of contraction when applied to the muscles. (This, however, only holds, strictly speaking, for percutaneous faradisation; exposed muscles or muscles excited by electrical acupuncture exhibit for a long time feeble contractions limited to the fasciculi directly excited.)

As in nerves, so in muscles, the loss of faradic excitability may persist for a long time, and in incurable cases forever. The faradic excitability of muscles returns after the commencement of regeneration and restoration of motility—usually somewhat later than in the nerves. With progressive recovery it also increases, though extremely slowly, and usually remains for a very long time at a low ebb, which is the more marked the longer the duration of the paralysis.

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[1] Ueber die Verschiedenheit der Aufnahmsfähigkeit und Leitungsfähigkeit im peripheren Nervensystem. Zeitchr. f. rat. Med. 3 Reihe, Bd. 29, S. 221.

[2] Versuche über intermitt Nervenerregung. Pflug. Arch. d. Physiol. Bd. VI. p. 180.

The *galvanic excitability of muscles* exhibits essentially different relations; during the first week it falls uniformly with the faradic, but in the course of the second week occurs a very remarkable augmentation of galvanic excitability which increases in the following weeks and is connected with qualitative changes, both in the order and mode of contraction. The muscles then react to currents which are so feeble in intensity as to be entirely inoperative on healthy ones, distinct contractions frequently occurring on interruptions of the current produced by only two elements. These are essentially different from normal contractions, for whilst the latter are short and lightning-like, the former are *slow and protracted*, the contraction, even with currents of small intensity, passing into a muscular tonus, which endures throughout the whole period of the transmission of the current. This difference in the form of contraction between sound and paralyzed muscles may be especially demonstrated at points where such muscles lie in close proximity with one another and can be coincidently excited with one electrode, as happens, for example, in the muscles of the chin in unilateral facial paralysis.

Coincidently with the increase of excitability a progressively *increasing qualitative change of the law of muscular contraction occurs.* This is manifested by a gradual and strong increase of the anodal closing contraction, so that soon this equals the cathodal closing contraction (AnSZ=KaSZ), or may even, in some cases, be greater than it. The opposite obtains for the cathodal opening contraction (KaOZ); this increases relatively more than the anodal opening contraction, so that it soon equals and ultimately surpasses it, and there is consequently a complete inversion of the normal formula of muscular contraction in regard to the strength of the contractions. When the alteration has attained a certain degree, the opening contractions disappear and, according to Brenner, in the same proportion as the slowness of the contractions increases, and their capacity to react to currents of short duration (see below) diminishes—obviously because the opening stimulus is only of brief duration. The alteration lasts at this stage, though with variations in different cases, for three, six, or eight weeks, or even for a much longer period.

To this succeeds, again, a gradual diminution of the increased galvanic excitability, whilst the qualitative changes of the excitability, and especially the preponderance of the anodal closing contraction, and the slow mode of contraction, still persist. By degrees stronger and still stronger currents are required to produce contraction, and in incurable cases, where marked atrophy, and finally the complete disappearance of the transversely striated tissue occur, an extremely feeble anodal closing contraction is usually the last indication of life which can be obtained from the vanishing muscle. If, on the other hand, regeneration and recovery take place, the normal mode of reaction is gradually re-established, while the galvanic excitability diminishes; but the excitability falls far below its normal degree, so that at a certain period of the paralysis the muscles may present a greatly diminished galvanic, with an increased faradic excitability. Moreover, a diminution of the galvanic excitability may be demonstrated for a long time after recovery has set in, especially in serious and protracted cases.

This depressed condition of excitability in muscle occurs, upon the whole, tolerably independently of the regeneration of the nerves and the restoration of motility; that is to say, it is not immediately connected with these, but requires a certain time for its course, so that at a period when the regeneration of the nerves is already actively proceeding, the changes in the excitability of the muscles may still persist without diminution. From this it results that in particular cases, according to the earlier or later occurrence of motility and regeneration, and the associated return of excitability in the nerves, a very different and apparently complicated aspect is presented by the electrical excitability of the nerves and muscles. If the regeneration sets in early, and the changes in the galvanic excitability of the muscles are still fully developed, it may happen that the abnormal galvanic reaction of the muscles may coexist with restored faradic and galvanic excitability in the nerves; it may happen also that, after recovery of the motility, the muscles may be excited to action by the nerves, in accordance with the ordinary law of contraction, although when direct galvanic stimulation is applied a qualitative change of the formula of contraction and of the mode of contraction may be observed. If, on the contrary, regeneration sets in late, it may happen that a (restored, but still) diminished electrical excitability of the nerves may coincide with a likewise diminished but still qualitatively altered galvanic excitability of the muscles. If, however, we keep in mind the regular course of the phenomena presented by both the nerve and the muscle, and take into consideration also the earlier or later occurring process of regeneration and its effects upon the excitability, we shall always be able to decipher and estimate correctly the symptoms that may be presented. I give below two tables of curves which represent graphically the possible combinations of these relations in regard to voluntary, faradic, and galvanic excitability, and their relations in point of time to the histological changes, and I trust they will faciliate the understanding of the numerous and diversely modified cases that are met with in practice.

The extraordinary fact that in the "reaction of degeneration" the muscle fails for a long time to react to the stimulus of the faradic current, whilst it readily responds to the galvanic stimulus, has been explained by Neumann in the following satisfactory manner. Muscles, he says, that have undergone this pathological change, have simply lost the power of responding to currents of momentarily short duration, whilst they react in an increased and qualitatively altered manner to currents of longer duration. But inasmuch as faradic currents are without exception currents of only momentary duration, the muscles do not react to them. If through muscles that contract freely on the application of an extremely feeble current of some duration, a very strong current be passed, to which some mechanical arrangement gives a merely momentary duration, no reaction will take place. Such is the physical explanation of the difference between the faradic and galvanic excitability of such paralyzed muscles; the value of the change by which the muscular tissue loses its normal power of reacting to currents of short duration is a question the solution of which must be left to the physiologists.

The *augmented excitability of the muscles to mechanical irritation* that occurs in the different paralyses is deserving of a few observations. It was discovered and described almost at the same time by myself [1] and Hitzig,[2] and has since been repeatedly observed. It consists in the circumstance, that the paralyzed muscles respond to slight mechanical stimulation (tapping with the tip of the finger, or a blow with a light percussion hammer, or even the removal of a body pressing upon them) by a distinct, slow and protracted contraction. The increased mechanical excitability in the muscles usually occurs at a somewhat later period than the augmented galvanic excitability; it generally increases rapidly, and is particularly well-marked in muscles having a firm, bony support; it then gradually diminishes and ultimately disappears in the course of the third or fourth month. It appears to be more or less connected with the increased galvanic excitability of the muscles, and to have the same significance, even if it do not run a perfectly parallel course with it.

When the cycle of changes just described has run its course, a considerable diminution of excitability, both for faradic and galvanic currents, without remarkable qualitative changes, generally remains both in the nerves and in the muscles. This often continues for a long time after apparently complete recovery of motility, though of course much longer when the recovery is incomplete. If such cases come under examination in their later stages, the still remaining changes in the electrical excitability afford no indications of the great varieties presented in the earlier periods of the affection.

It is a point of great practical importance that all these symptoms *stand in the closest relation with the histological changes in the nerves and muscles* that have been above described *in extenso;* the presence, therefore, of the *reaction* of *degeneration* announces the existence of these histological changes, and from the stage of the change in the excitability an opinion can be formed of the stage to which the histological change in the nerve and muscle has advanced. This connection may be formulated as follows :

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[1] Verhandl. d. Heidelberg. naturhist-med. Vereins, Band IV., p. 116. Heidelberg. Jahrbüch. Juni. 1867.

[2] Ueber die Mechan. Erregbarkeit gelähmter Muskeln. Virchow's Archiv., Band XLI., p. 301. November, 1867.

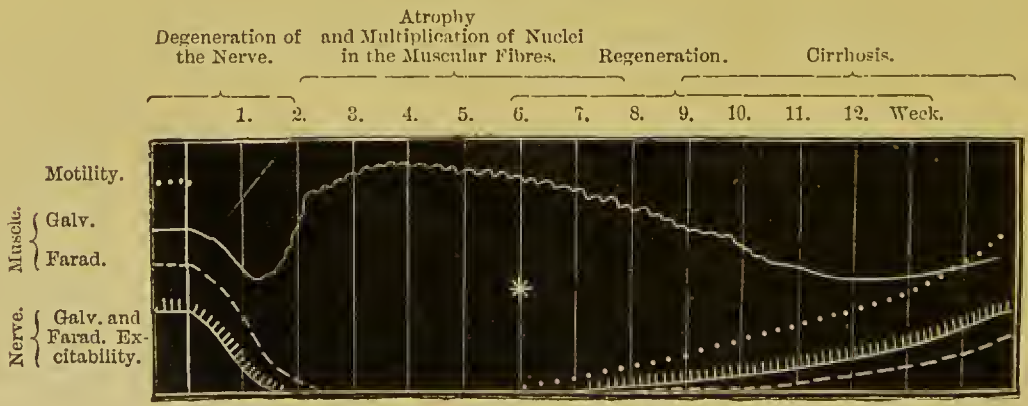
The degeneration in the *nerve* corresponds precisely in point of time to the diminution and extinction of the electrical excitability of the nerves; if the degeneration has reached a certain degree, the nerve is no longer excitable either by galvanic or by faradic currents. But as soon as regeneration commences, and a connection is established between the centric and peripheral nerve segments, motility begins to be restored. As the process of regeneration extends along the fibres in the peripheral portion of the nerve, the excitability of this portion also begins to return, and it steadily increases with the increasing breadth and development of the regenerating fibres. It remains, however, for a variable period below the normal amount, partly on account of the considerable hypertrophy of the connective tissue in the nerve itself, but partly also and chiefly owing to the atrophy and cirrhosis of the muscles, which of course are incapable of contracting with the same energy as healthy muscles under the influence of the same irritation.

The primary diminution of electrical excitability in the muscles does not correspond with any demonstrable changes in the muscular substance itself; it must therefore be referred to the progressive degeneration of the intramuscular branches of nerves and their terminations, and therefore pursues a parallel course with the diminution of excitability in the nerves. Complete extinction of the excitability does not occur even when the nerves are completely degenerated, because the muscular substance, with its specific irritability, still remains. The histological and chemical changes, which are so distinctly perceptible in the course of the second week in the muscular substance, correspond to the well-marked increase and qualitative changes of the galvanic excitability. The advance of anatomical changes, and especially of the atrophy of the muscular fibres, corresponds to the subsequent diminution of galvanic excitability ; the compensation of the nutritive disturbance in the muscular fibres during healing, to the return of the normal mode and law of contraction. Lastly, the well-marked hypertrophy of connective tissue in the muscles, and the advanced atrophy of their fibres, explain why a condition of considerably diminished excitability long remains even after complete restoration of the motility.

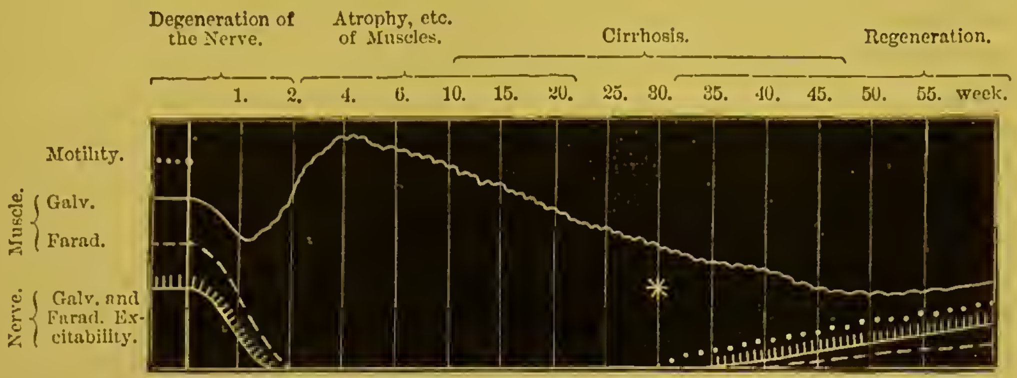
I have attempted to illustrate, graphically, all the foregoing states and relations, and give three tables of curves, in which the successive alterations in the electrical excitability of the nerves and muscles are presented, as well as the relations that exist between the motility and the histological processes for three categories of cases (one of rapid recovery, one of slow recovery, and one incurable case). The various relations of motility, excitability, and histological characters, which are present in the different stages of paralysis, can here be seen at a glance,—a point of no little practical importance. Of course, these curves—on account of the want of space—lay no claim to absolute exactness; they only give a general view of the relations, and require but little explanation.

The first thick vertical line, or ordinate, indicates, in all the drawings, the attack of paralysis, the sudden cessation of the motility (•••); the period of the return of motility is indicated by a star (*). The succeeding ordinates represent intervals of one or more weeks, dating from the occurrence of the attack. The undulations in the line representing the galvanic excitability of the muscle indicate its qualitative changes. Table 1, for instance, exhibits the diminution of excitability that occurs, both in nerve and muscle, during the first week; the extinction of excitability of the nerves and of the faradic excitability of the muscle, the augmentation and qualitative change in the galvanic excitability of the muscle in the second week; and the return of the motility in the sixth week. In the eighth week it may be seen that the motility has been restored to some extent, that the nerve has recovered its faradic and galvanic excitability, and that there is an increase and qualitative change in the galvanic excitability of the muscle, and so on. At the same time the stage of histological change in the muscle and nerve may be deduced from what has been above stated.

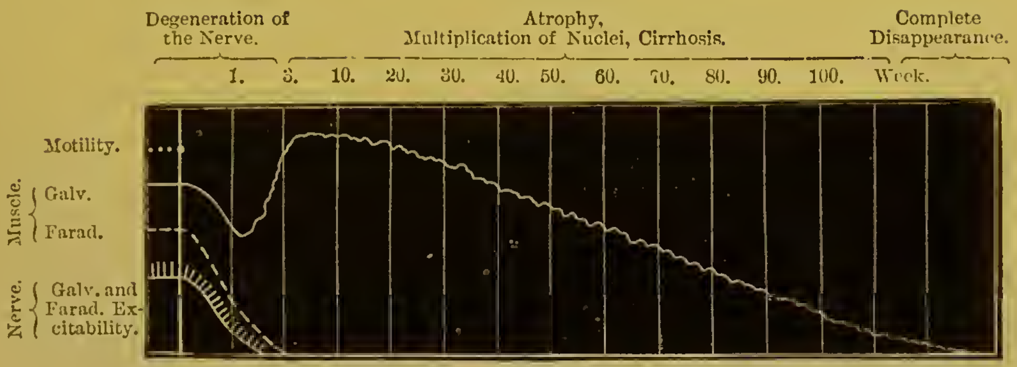
1. *Recovery Rapid.*



2. *Recovery Slow.*



3. *No Recovery.*



A few brief observations in regard to the more subordinate relations may be added to the foregoing representation of the "reaction of degeneration," which has been drawn from many cases, and is intended to represent the general type and character of the reaction.

Deviations from the typical characters of the reaction of degeneration are not uncommon, and of these it is difficult to give any explanation, though when accurately followed, they may perhaps prove of importance in solving the problem of trophic influence; thus, for example, the usual inexcitability of the nerves may exist, yet without the well-marked increase of the galvanic excitability of the muscles, which has either not occurred at all, or has been only transient. The qualitative changes of the contraction are, however, usually present. This occurs chiefly in the lower extremities, and apparently also in the spinal paralyses of children, in which, however, examination is very difficult to make, and, for the most part, is only practicable in the later stages.

It is further observable that the diminution in the faradic and galvanic excitability of the nerves is quite inconsiderable, whilst the muscles exhibit a very high degree of galvanic and mechanical excitability, in addition to qualitative changes,

—a condition that may persist for some time after recovery. The first cases of this kind that came under my notice (which were almost exclusively cases of facial paralyses) I endeavored to explain by referring them to the early restoration of the conductivity and electrical excitability of the nerves. In cases, however, that I have had the opportunity of following throughout their whole course, I have distinctly ascertained that only a slight diminution of electrical excitability occurred in the nerves whilst the galvanic excitability of the muscles underwent its ordinary and characteristic changes. This indicates the occurrence of progressive anatomical changes in the muscles *without* complete degeneration of the nerves, and a smaller degree of disturbance of nutrition in the nerve, with a tendency to prompt recovery. In all these cases the course of the paralysis till complete recovery had taken place was relatively rapid.

Unimportant variations in the time of the appearance and in the succession of the several characteristic symptoms of course occur, and are usually referable to accidental circumstances (unusual intensity or extent of the cause of the paralysis, volume and direction of the muscles, etc.).

The very different behavior of the nerves and muscles has not of late been sufficiently attended to. Cases are constantly reported in which it is lightly stated that the *nerve* was inexcitable by faradic stimulation, but reacted in a qualitatively changed manner to the galvanic current,—that is, behaved as only the muscles usually do. Such statements probably all rest on defective observation and on the adoption of inefficient means of protecting the muscles from the effects of conduction, and they can only be accepted as correct where it is shown that due precautions have been taken against error.

It does appear, however, that such cases are sometimes met with under exceptional conditions. I have myself observed it once in the nerve of a frog, and Cyon on one occasion in the nerve of a rabbit. Like Brenner, I have never observed it in man. It must consequently be of very rare occurrence in practice, and more definite statements based on careful investigations are much to be desired on so interesting a subject.

Very recently Vulpian has opposed the statements made by myself, Ziemssen, and Weiss, in regard to the galvanic excitability of the *muscles*. He has not found an increase in their galvanic excitability, nor preponderance of the anodal closing contraction over the cathodal closing contraction to be of constant occurrence. The reason of this is, perhaps, that Vulpian's researches were not undertaken methodically, but were only made in those stages of the paralysis in which the change had either not yet made its appearance (during the first and second week), or when it had passed by (from the eighth to the tenth week). The most important particulars are lacking. In truth, I know no alteration which can be found with greater certainty and distinctness than the quantitative increase and the qualitative change of the galvanic excitability of the muscles in traumatic paralyses. I have found and demonstrated this in so large a number of cases, and it has been corroborated by so many other observers, that I must maintain, in opposition to Vulpian, the correctness of the above statements.

I am unable to assign any weight to the evidence adduced by Vulpian against my view in regard to the anatomical basis of the difference in the conductivity and excitability of the regenerating nerves. On the strength of the above-mentioned clinical and histological facts, I have expressed the opinion that the axis cylinder is subservient to conduction, and the medullary sheath to the reception of excitations, but must leave the substantiation or refutation of this view to experimental physiology. For practical purposes it was sufficient to have experimentally demonstrated the anatomical and physiological basis for the remarkable behavior observed in pathological cases. I find, moreover, in Weir Mitchell[1] an observation which seems to be in favor of my opinion; in the nerves of a rabbit, which had been exposed to a paralyzing pressure, but which had so far recovered from its effects that conduction of stimuli through the compressed part was restored, microscopical investigation showed that the medullary sheath was in the same state of change as in a nerve six or seven days after section.

The "reaction of degeneration," to which I have given this name on account of its intimate connection with the degeneration of the nerves and muscles, appears—just as in experimental paralysis—in all *paralyses of traumatic origin* accompanied by complete division or crushing of the nerves; it also occurs in certain paralyses of rheumatic origin (and with special frequency in facial paralyses), probably when the cause of the paralysis has produced severe compression of the nerves. Under the same circumstances it also occurs in paralyses from *neuritis*, in compression of the nerves by *tumors*, *extravasations of blood*, *retraction of cicatrices*, and, in fact, wherever peripheral nerves are severely injured mechanically, in whatever way this may happen. It occurs also in *saturnine paralysis*, and in the so-called *spinal paralyses of children*, in which it is usually limited to particular groups of muscles. Lastly, it occurs in the very obscure group of "paralyses of the acute diseases," which are partly equivalent to peripheral paralyses from compression, and are partly spinal paralyses, analogous in their character to the spinal paralyses of children.

From the description just given it is easy to discover what positive conclusions can be drawn from the existence of the reaction of degeneration. Thus we may conclude with positive certainty that *wherever this reaction is presented, considerable anatomical changes must exist in the nerves and muscles*, the exact nature of which may be deduced with some degree of precision from the stage to which the electrical changes have advanced.

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[1] Weir Mitchell, Injuries of Nerves, p. 113.

The second conclusion that may be drawn, though not always, perhaps, with certainty, is that *where the reaction of degeneration is present, the case is one of peripheral paralysis.* Two exceptions only are at present known to this rule: lead paralysis, in regard to which we are ignorant whether it is of peripheral or centric origin; and the spinal paralysis of children, which has of late years been very generally regarded as of spinal origin. The facility, however, with which these two forms of disease can be diagnosticated by their many characteristic symptoms, so that, as a rule, electrical investigation is unnecessary to establish the diagnosis, renders the above rule one of wide application. The (rare) cases of spinal paralysis in adults, which are analogous to the cases of spinal paralysis in children, may be easily recognized, from the other symptoms, as being of spinal origin. Besides these well-marked exceptions, the reaction of degeneration is of great diagnostic value in establishing the distinction between peripheral and centric paralyses, as, for example, in many basal paralyses of the cerebral nerves, in rheumatic paralyses of particular cerebral nerves, in the paralyses of nerve trunks within the spinal column, etc. The details will be given when the special forms of paralysis are under discussion.

It is scarcely possible to make any general observations in regard to the *course* of paralysis. It is already obvious, that, from the great number of the causes of these affections and the great diversities in the seat, the course may vary exceedingly. Thus, the commencement of the paralysis may be either quite sudden or more gradual; in the latter case it may progress equably or by fits and starts, the variation being almost entirely the result of the nature of the cause. The progress of the paralysis usually takes place from the most remote parts towards the centre, proceeding, especially in spinal paralysis, steadily from below upwards. In cerebral paralysis, the arm is frequently first affected, and then the leg and face. When the paralysis has once developed itself, it may either remain stationary, or it may gradually go on to recovery, or it may undergo considerable variations, improvements, or exacerbations. The great uniformity in the course of many paralyses is not unfrequently interrupted by the occurrence of symptoms of motor irritation, by the alternate appearance and disappearance of sensory disturbances, and by the occurrence of trophic disturbances. The detection of successive changes in the electrical excitability serves to enliven the monotony of the disease. These, however, are all circumstances liable to very manifold modifications, according to the kind, place, intensity, extent, and progressive or retrogressive course, of the cause of the paralysis, and consequently cannot here be discussed in detail.

The *duration* of an attack of paralysis is as variable as its cause. Some paralyses last only a few minutes or hours (as in the case of slight paralyses from pressure, ischæmic paralyses), whilst others persist for a few days or weeks (slight rheumatic paralyses, toxic paralyses); there are others, again, which never proceed to recovery before many weeks or months have elapsed, such as severe traumatic or rheumatic paralyses, the majority of cerebral and spinal paralyses, paralyses from lead, etc.; lastly, there are others from which recovery never takes place, and which continue through life, as in all those forms in which recovery from the cause is anatomically impossible.

The *terminations* of paralysis depend, for the most part, on the cause, the seat, and the more or less progressive character of the disease. Recovery occurs in many cases, principally as the result of restoration of the conductivity and excitability of the motor apparatus, partly also in consequence of the removal of the inhibiting cause, and partly from regeneration of the motor nerves themselves. That these processes are possible in many cases, in traumatic, rheumatic, neuritic, meningitic and myelitic paralyses, in apoplectic, toxic, and reflex paralyses, and in paralyses from compression, is taught by observation and experiment, and is fully corroborated by daily experience. No further evidence is required to prove that recovery sometimes occurs slowly, and sometimes with rapidity, and that frequently traces of the disturbance are recognizable for a long period. Recovery may also occur by collateral motor nerves gradually assuming the function of the injured ones, acting as it were vicariously for them, and by constant exercise almost restoring the normal state of functional activity. It is highly probable that recovery is effected in this way in certain forms of cerebral and spinal paralyses, and the more so, since there are both anatomical and physiological grounds for believing in the existence of numerous connections between motor nerves ; it is obvious that in many such cases *incomplete* recovery will alone take place. This is indeed a very common termination of paralysis ; the muscles only recover a part of their strength and functional activity, their contractions continue to be slow, stiff and awkward, and are often limited by contractures ; certain of them never recover their motility at all ; in short, innumerable forms of defective motility may persist, the degree of which is determined essentially by the cause of the paralysis and the possibility of its removal.

Lastly, death may be directly caused by the extension of the paralysis to parts necessary to life, especially to the respiratory apparatus and the muscles of deglutition, or indirectly by the formation of bed-sores, the development of serious lesions of the bladder, etc., which lead to a fatal termination in the ordinary way. Death must not, however, be attributed to the paralysis when it is occasioned by the effects of a lesion which has accidentally produced the paralysis, as, for example, cancer, tuberculosis, and lead in toxic doses.

Diagnosis.—The diagnosis has to extend over several points, and is of the greatest importance in determining the proper measures to be adopted in the treatment, since this depends essentially upon a correct appreciation of the seat and kind of the cause of the paralysis, and of the nature of the consecutive changes.

It is self-evident that a precise diagnosis can only be made after a careful and complete investigation of all the circumstances of the case, and whilst we must refer the reader to what has been already said (p. 267) in regard to the examination of the motor organs, we must add that a test of the sensibility and of the degree and extent of its impairment must always be made. It is important also that any disturbances in the evacuation of the bladder or bowels, in the movements of the intestines and uterus, in the power of erection, sexual power, etc., should not be overlooked. Lastly, the state of the intellectual powers and the activity of the sensory organs may be investigated. It is only by such a full and profound examination that a complete picture of the disease can be obtained, though it is not of course necessary in all instances, and in local peripheral paralysis, for example, the examination may be almost limited to the local changes. In all difficult and complicated cases, however, it is by a complete investigation alone that a reasonably accurate diagnosis can be made.

The examination must, in the first instance, be directed to the question, whether paralysis is certainly present. This is generally easily ascertained by the circumstance that when an effort of the will is made, no muscular contraction follows. We may, however, still find ourselves in some difficulty; the paralysis, for example, may affect muscles that never act independently, and whose physiological action has not been clearly determined. In such a case it is impossible to arrive at any positive conclusion, a matter, however, of small importance. The determination of the existence of paralysis is also difficult in those who are unconscious and incapable of making any effort of the will, as, for example, in apoplexy; but even here the paralyzed side may, in general, be distinguished by its appearing, as a rule, much more flaccid than the other, by its exhibiting no involuntary movements, and no movements during delirium, etc. It must be borne in mind that motor disturbances resembling those of paralysis may be caused by ataxy and by disorder of the muscular and cutaneous sensibility.

The diagnosis of the *seat* of the paralysis, or, in other words, of the part of the motor apparatus affected by the paralyzing lesion, is of the greatest importance. For the determination of this point, various aids to research and methods of investigation must be adopted. In the next place we have to decide whether the paralysis is *neuropathic* or *myopathic*. This, as a rule, is not very difficult to determine, since in myopathic paralyses the lesion usually commences in particular muscles, and gradually spreads to others, frequently even from one muscular fasciculus to another; other points aiding in the differential diagnosis consist in the atrophy preceding complete paralysis, in the occurrence of fibrillar contractions and pain in the muscles, in the diminution of the electrical excitability, pursuing an exactly parallel course with the diminution of the volume of the muscle, and, lastly, in the presence of a demonstrable local cause.

It is practically more advantageous in neuropathic paralyses not to distinguish between paralyses of central origin and paralyses of the conducting apparatus (nerve fibres), but to determine the seat of the paralyzing influence, and to decide whether we have to deal with a *peripheral paralysis* (the seat at any point in the course of the nerves after their emergence from the brain or spinal cord), or with a *spinal* paralysis (caused by disease of any segment of the spinal cord), or with a *cerebral* paralysis (resulting from disease of the brain). The principal points on which we must rely for distinguishing these three groups of paralysis will be briefly stated.

Peripheral paralyses are probably always paralyses of conduction, the causes of which may affect the motor nerves at any point between that of their emergence from the brain or spinal cord and that of their entrance into the muscles. They present the following characteristics. They are limited to the region supplied by one or a few nerve trunks; this is of especial importance in the extremities; exceptions occur when the seat of the paralysis is in the orbit, at the base of the skull, or in the lower part of the spinal canal where the cauda equina contains numerous nerve trunks, on which account the extent of the paralysis is increased so that it may even present the form of paraplegia. The non-participation of branches of the same nerve springing from a higher plane is also in favor of the peripheral origin of the affection. Anæsthesia is almost always coincidently present, and is precisely limited to the region of distribution of the mixed nerves affected. There is never, however, any retardation in the conduction of sensation (Weir Mitchell). In addition to the voluntary movements the reflex automatic and associated movements also are completely absent. Spasms occasioned by central disease do not extend to the paralyzed muscles. *Marked* vaso-motor and trophic disturbances, especially an early and well-marked atrophy of the muscles, are circumstances in favor of the peripheral origin, and the same may be said of the presence of the reaction of degeneration on electrical investigation (of course with the above-mentioned two exceptions); complete preservation of the electrical excitability is not opposed to a peripheral seat of the cause of the disease. Electrical testing, especially if it be possible to apply the stimulus above the seat of lesion, may often materially aid in determining its seat; if no reaction takes place on excitation of the central segment of the nerve, whilst it is easily produced when the peripheral segment is excited, the seat of the cause of paralysis must lie between these two points, and must consequently be peripheral. Lastly, in peripheral paralysis no morbid phenomena indicating disease of the spinal cord or brain are present, though accidental complications with central diseases may lead to errors which can only be avoided by the greatest caution. Even the existence of a peripheral cause (as of a wound) is not always decisive evidence in favor of the peripheral seat of the lesion, because the wound sometimes also produces coincident central lesions; thus, for example, it is not always easy, in wounds of the skull, to determine whether the injury has affected the nerve trunks at the base of the skull or the motor fibres within the brain.

The characteristic feature of *spinal* paralysis is the occurrence of a nearly uniform ascending paralysis of both sides—paraplegic—though this is not absolute proof of its spinal origin, since similar cases have been met with arising from paralysis of the cauda equina or even from some lesion of the brain (cerebral paraplegia!). In accordance with the height which the disease has reached in the spinal cord (lumbar, dorsal, or cervical region), the symmetrical groups of muscles belonging to the lower extremities, trunk, belly, and upper extremities, are paralyzed progressively from below upwards. Disturbances of sensibility are very common, though they are not always equal in extent and intensity to those of motility; they are often limited to abnormal sensations (numbness, formication, etc.) in the feet, and retardation of the conduction of sensation is frequently present. Pain in the back, and a sensation of constriction around the body (Gürtelgefühl), are of common occurrence. Sensory disturbances are always absent in the spinal paralysis of children and the allied forms of disease. Disturbances in the functions of the bladder (incontinence and retention of urine) and of the sexual organs (priapism, pollutions, spermatorrhœa, and impotence) are amongst the most characteristic symptoms of spinal paralyses. Some of these, however, may also make their appearance in peripheral paralyses of the cauda equina. Paralytic myosis often occurs in affections of the cervical region of the cord. Reflex movements are for the most part preserved, and are often increased in intensity; occasionally, when there is uniform degeneration of the whole medulla, they are abolished. The automatic movements may, according to the part of the cord affected, either remain unaffected or participate in the paralysis.

Convulsive movements proceeding from the brain, as, for example, in epilepsy, do not extend to the paralyzed parts; on the other hand, muscular tensions and spasms proceeding from the spinal cord are not unfrequently seen in these parts. Vaso-motor and trophic disturbances, as we have above pointed out, are not of unfrequent occurrence, but, at the same time, are not constant. Psychical affections, and affections of the organs of special sense, and of the cerebral nerves, are usually absent; experience teaches, however, that in certain forms of spinal disease (especially tabes) affections of the optic nerves, and of the nerves distributed to the muscles of the eye, are extremely common; these cases, however, are for the most part easily recognized. No very positive conclusions can be drawn from electrical testing, though it is probable that some spinal affection (especially tabes) is present, if, after the disease has lasted some time, there is a moderate increase of the excitability of the paralyzed parts. So, too, simple diminution of electrical excitability, without qualitative change, but accompanied by moderate atrophy of the muscles, is in favor of a spinal affection, since this condition does not readily occur in peripheral paralyses, and scarcely ever in those of cerebral origin. In many cases, finally, no conclusions can be drawn from the application of electrical tests.

We have still to mention that in exceptional cases hemiplegia may occur from disease of the spinal cord, as in the so-called unilateral lesion (Brown-Séquard). In such cases there is usually sensory paralysis of the opposite side, with motor paralysis of the same side as the spinal lesion. The lower extremity is usually alone affected, though if the seat of the lesion be high, the upper one may be also implicated. A correct diagnosis may usually be made by the freedom from impairment of the facial muscles, the absence of all cerebral symptoms, and the presence of vesical disorders.

The symptoms present much greater diversity in *cerebral paralyses*, rendering their characteristics more complex and the diagnosis more difficult. In ordinary well-marked cases, as in those proceeding from extravasation of blood, embolism, tumors, etc., hemiplegia occurs upon the opposite side of the body, the arm being usually most affected, and the face and tongue least. At the same time cerebral disease may be the cause of many isolated forms of paralysis; thus the paralysis may be limited to particular nerves, plexuses, or trunks, and it may be variously combined and complicated with all possible disturbances. This is no doubt attributable to the circumstance that in various parts of the brain different motor nerves lie in close contiguity, so that as the seat of the lesion varies, the grouping of the symptoms is different, whilst, on the other hand, in consequence of the separation of the various motor paths in the brain, it often happens, that when the lesion is circumscribed, the paralysis may be limited to special muscles or groups of muscles. The diagnosis can hence only be rendered tolerably certain by a thorough knowledge of the anatomy and physiology of the brain, combined with some experience in its diseases. In cerebral paralyses, reflex actions are almost always preserved, and frequently increased in energy ; associated and automatic movements are usually unaltered, the former being also often increased. Symptoms of motor irritation (contractures, twitchings, and spasms) are not unfrequent in the paralyzed parts, and are often more strongly marked in them than in the healthy parts ; epileptic convulsions, and those caused by strychnine, occur also in the paralyzed parts, and are often more violent there than in the healthy ones. Disturbances of sensibility are both more rare and less marked than in peripheral and spinal paralyses; they are often present at the commencement of the attack, but usually soon disappear. Vaso-motor disturbances are very common, but trophic disturbances are very rare; atrophy of the muscles in particular scarcely ever occurs (except in paralysis of the pons). Disorders of the functions of the higher organs of sense, of the different cerebral nerves, and especially of the intellectual powers, are highly characteristic, and of considerable diagnostic importance, the last-named symptom in particular being scarcely ever absent, though it may not become very prominent. With them may be enumerated the not unfrequent occurrence of disturbances of speech, whether intellectual (aphasia) or peripheric (alalia). Alterations in the size of the pupil, headache, vertigo, vomiting without apparent cause, are also symptoms of great importance. Electrical investigation supplies much information. Slight and uniform increase in the excitability speaks in favor of the central origin of the paralysis, as does also a normal reaction when the disease has been of very long duration. Distinct and strongly-marked diminution occurs only in paralysis resulting from disease of one of the peduncles of the brain, which, however, is easily recognized by other symptoms.

Cerebral *paraplegiæ* are very rare, and generally occur in the form of two separate hemiplegiæ, one side being more severely attacked than the other. In such cases the diagnosis is much facilitated by the coexistence of psychical symptoms, by the implication of various cerebral nerves and of the organs of special sense, and by impairment of speech, etc. We need not here dilate upon the local diagnosis of cerebral paralyses, but must refer to the account of cerebral diseases given in another volume of this series. We may, however, remark that it presents great difficulties from the complexity, on the one hand, of the physiological paths, and, on the other, of the pathological processes usually present in these affections, since, in addition to the primary lesion, compression and degeneration of the nerve fibres at the seat of the disease, secondary hyperæmia and stasis, œdema, and marked pressure on the brain, etc., may occur. To form an accurate diagnosis, under such circumstances, is one of the most difficult tasks in the whole range of pathology.

Numerous cases of paralysis also occur where the paralyzing lesion may affect several distinct parts, so that paralyses of peripheral and spinal, of spinal and cerebral, and of peripheral and cerebral origin, may coexist. In such cases a correct conclusion in regard to the nature of the disease can only be arrived at after exact research and much consideration.

The diagnosis of the localization of the paralysis is, however, not enough; it is further necessary to determine *what kind of lesion* is present; in other words, what is the nature of the cause of the paralysis—whether it is due to a wound, to inflammation, to neoplastic formations, or to rheumatism; whether degeneration, extravasation of blood, poisoning, or some reflex disease be present. This presents immense difficulties, and in some cases is quite impossible; particular attention should, however, be paid in all instances to the anamnesis, the entire aspect of the disease, its course, and the results of electrical research, in order to obtain the necessary insight. General statements can scarcely be made, and the points required for diagnosis will be given in the description of the several forms of paralysis.

We have here only shortly to characterize particular forms of paralysis in which we are still in doubt respecting the seat and the nature of the primary lesion, but which may be tolerably well distinguished from other paralyses by a whole series of symptoms; but even with regard to these forms, the precise details must be left to the special part and to the various sections of this work specially relating to them.

The diagnosis of *hysterical paralysis* rests essentially upon the existence of other well-marked hysterical symptoms; but at the same time it must not be forgotten that even hysterical patients may occasionally have a rheumatic, traumatic, neuritic, or other form of paralysis. True hysterical paralyses may, protean-like, occur sometimes here, sometimes there, affecting particular nerves and plexuses, and presenting even the form of paraplegia or hemiplegia; they may rapidly disappear, or be extraordinarily obstinate; their appearance and disappearance are not unfrequently occasioned by violent emotion, by an attack of hysterical convulsions; generally speaking, a high degree of anæsthesia is present; the electrical excitability of the muscles is perfectly preserved, while the electro-muscular sensibility, according to Duchenne, undergoes considerable diminution. The impression is frequently given that the patients only lack the ability to direct the influence of their will upon the paralyzed parts. All indications of serious organic lesion of the brain and spinal cord are absent; the patients never develop bedsores, and paralysis of the bladder, if present at all, is only so in a very inconstant manner.

Diphtheritic paralysis is characterized and readily recognized, even in cases in which the antecedent pharyngeal or other form of diptheritis has not been certainly known to exist, by the sequence and the peculiar combination of the symptoms of paralysis. Its commencement with paralysis of the velum palati, nasal twang in the speech, difficulty in swallowing, the occurrence of mydriasis, paralysis of accommodation, and pareses of the muscles of the eye, the remarkable retardation of the pulse, the progressive pareses and paralysis of one or all four extremities, collectively constitute an extremely characteristic picture of disease. The sensibility is only moderately disturbed; the bladder and rectum are for the most part unaffected; electrical investigation frequently exhibits the reaction of degeneration in the velum palati (Ziemssen and others), and frequently simple diminution of excitability; whilst there are no characteristic changes in other muscles and nerves.

Lead paralysis may usually be recognized with the greatest facility by the fact that very definite groups of muscles are successively affected. The extensor muscles of the forearm, those supplied by the radial nerve, are first attacked, whilst, which is a very characteristic symptom, the supinator longus escapes. The morbid process then extends to the small muscles of the hand and the flexors; in the lower extremity the muscles supplied by the peroneus are most often affected. The presence of well-marked atrophy of the muscles, the existence of the reaction of degeneration, and ultimately extreme diminution of electrical excitability, may be considered as evidence confirmatory of the diagnosis. Sensory disturbances, as a rule, are entirely absent.

The diagnosis will be rendered still more certain, though we may not always succeed in discovering the source of the poisoning, if, in addition, other well-known signs of lead intoxication, such as the blue line on the gums, attacks of colic, saturnine arthropathies, cachexia, etc., are, or have been, present.

In regard to the *prognosis* of paralysis only a few observations of a general nature can be made, since it must always depend upon the particular circumstances of each case. Many forms of paralysis, such, for instance, as slight rheumatic facial paralysis, hysterical paralysis of the vocal cords, various paralyses of the ocular muscles, and the ordinary paralysis of the musculo-spiral nerve from pressure, may be classed among diseases which are easily and certainly curable ; on the other hand, there are others which are always severe and protracted affections, which occasionally, in spite of every effort to cure them, progress steadily to a fatal issue, or at least remain stationary throughout life. Upon the whole, the prognosis must, in a majority of cases, be given very guardedly, for it only too frequently happens that, even in apparently favorable cases, debility, paresis, in short, a deficiency in the functional activity of the affected part, remains permanent.

Some observations may, however, here be made indicating the points on which the general prognosis of paralyses may be founded. In the first place, it must always rest essentially on the nature of the cause of the paralysis. The forecast is worst in those cases where considerable portions of the motor nerves are completely destroyed by wounds, effusions of blood, degeneration, softening, etc. ; then again, in such cases the prognosis must be still further modified by the extent of the part injured, and by the number and nature of the accidental circumstances which may interfere with the reunion or regeneration of the divided ends of the nerve. Baerwinkel[1] has suggested a symptom of considerable prognostic value in severe traumatic paralyses, namely, that if, in the earlier stages—that is, in the first few months—pressure on the nerve below the point of injury is followed by an excentric sensation, a favorable anticipation may be formed in regard to the restoration of motility (because either the continuity of the nerve trunk was not completely interrupted, or regeneration of the sensory nerves has already commenced). The prognosis is less unfavorable in those cases which are due to simple compression, especially when the compressing body can be removed ; even then, however, recovery is often very slow.[1]

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[1] Arch. d. Heilk., 1871, p. 336.

A favorable prognosis may in general be formed of rheumatic and toxic paralyses, though the duration of the disease is often considerable. Recovery usually takes place from paralyses after acute diseases, especially diphtheria. The prognosis of hysterical and the greater number of syphilitic paralyses is decidedly favorable.

The *localization* of paralysis, independently of the cause, is of relatively small importance in the prognosis, since curable and incurable paralyses occur in all parts of the motor nervous apparatus. It may, however, be broadly stated that, *ceteris paribus*, peripheral paralyses are less serious than central, and that amongst the latter the prognosis is more favorable in cerebral than in spinal cases. The prognosis of central paralyses is good or bad in accordance with the prognosis of the cerebral or spinal disease by which they are produced. The prognosis of myopathic paralyses must in general be regarded as unfavorable.

The results of electrical investigation are often of value in forming a prognosis. Normal electrical excitability has in some cases (rheumatic facial paralyses) a decidedly favorable significance ; whilst in those cases of the same form of paralysis, in which the reaction of degeneration exists, the prognosis will be unfavorable,—in fact, if the reaction of degeneration be well marked, the affection may always be expected to have a duration of at least several months. When every trace of electrical excitability, even the last trace of galvanic excitability in the muscles, is lost, the prognosis is absolutely unfavorable, and recovery is no longer possible. The prognosis is rendered unfavorable when strongly-marked vaso-motor and trophic disturbances are present, and, above all, by the presence of serious and extensive sloughing bed-sores. The more intense the accompanying anæsthesia, the less favorable the prognosis. Paralysis of the sphincters of the bladder and of the rectum, decomposition and alkalinity of the urine are bad symptoms. Commencing paralysis of the respiratory apparatus and of the muscles of deglutition generally indicates impending dissolution.

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[1] A very remarkable example of rapid restoration of motility in the radial nerve after it had been lost for sixteen months, by the removal of the compressing callus, is reported by Busch in the Berl. klin. Woch., 1872, No. 34.

General Treatment of Paralysis.

It is scarcely necessary to repeat, what has been already so frequently said, that in the treatment of paralysis it is necessary, in the first instance, to attend to the *causal indication;* for if the cause of the paralysis be irremovable or incurable, all direct treatment will certainly fail. We have seen in the section on the etiology of paralysis how manifold the conditions may be against which the causal treatment must be directed, and, were we to mention everything that might require attention, we should have to recapitulate a great part of the special treatment of the diseases of the brain, spinal cord, and peripheral nerves. This would be of course superfluous; and we shall therefore only give a few hints as to the mode and kind of causal treatment that should be instituted in the different forms of paralysis.

In *peripheral paralyses* the removal of all agents effecting compression, such as tumors, abscesses, exostoses, cicatrices, and the like, is of primary importance; and if this removal be unavoidably accompanied by resection of the nerve, the greatest care must be taken to place the divided ends in as favorable a position as possible for their reunion. In traumatic paralyses little can be done directly to favor union, beyond adaptation of the two ends, though in open wounds a suture may be passed through them, and the treatment must otherwise be limited to appropriate antiphlogistic measures. In very old and apparently incurable cases of traumatic paralysis of the extremities an attempt may be made to dissect out the ends of the nerve and bring them into apposition, an operation which is much facilitated by the bloodless method of Esmarch. Neuritic paralyses require the application of local antiphlogistics, derivatives, and galvanic treatment; and the same means are serviceable in rheumatic paralyses.

In *centric* paralyses attempts must be made to remove or cure the alterations that may have been caused by traumatisms, extravasation of blood, inflammation, hyperæmia, anæmia, degeneration, proliferation of connective tissue, sclerosis, and tumors of all kinds. On these points the reader is referred to the several sections on diseases of the brain and spinal cord. A great number of remedial measures may here prove very serviceable, such as the application of cold, abstraction of blood, derivatives of all kinds, and especially the application of galvanism to the brain and spinal cord. All those means from which a favorable action on the anatomical causes of paralysis may be expected, and which can effect the absorption of exudations, of apoplectic clots, and of hypertrophic connective tissue, as well as the cure of degenerative processes,—*e.g.*, iodide of potassium, mercurials, and baths of all kinds (warm and ordinary saline baths, sea-water baths, mud baths, cold water cures, vapor baths, and Roman baths)—may be occasionally employed, and may become extremely important and successful means of curing the paralysis. The special indication for the employment of these means can only result from an accurate diagnosis and from a consideration of the special characters presented by each case.

Paralyses resulting from an unhealthy condition of the blood require appropriate tonic and dietetic as well as medicinal treatment; in toxic paralyses the treatment must be primarily directed to the removal of the poisonous agent. In reflex paralyses the removal of the peripheral cause is not always immediately successful, but should be invariably attempted. Paralyses occurring after acute diseases require, as a rule, only such causal treatment as may be appropriate to the nature and seat of the causative affection (spinal affections, apoplexy, neuritis, etc.). In certain constitutional paralyses, on the other hand, the treatment of the primary disease (syphilis, rheumatism, scrofulosis) is urgently required, and frequently leads to brilliant results. In the majority of cases, however, causal treatment alone is insufficient, and after the removal of the cause, it is further necessary to improve the nutrition and hasten the excitability and conductivity of the motor nerves, in order that motility may be perfectly re-established. The remedies to be applied for this purpose are *immediately directed to the cure of the paralysis;* they nearly all aim at restoring the normal nutrition, excitability, and conductivity of the motor nerves, by strongly exciting them in a direct or reflex manner. They are applicable in all instances where no causal indication for treatment is present, or where it appears advisable to institute, in addition to the causal, direct anti-paralytic treatment.

We are at present only in possession of very few remedial means that may thus be employed with advantage. The most important and the most generally applicable is *electricity;* next to this is *treatment by baths*, and, in certain cases, gymnastics, described as the so-called gymnastic-cure. Direct anti-paralytic internal remedies, and an incredibly great number of external remedies, which, from time immemorial, have played so great a part in the popular treatment of paralysis, and also in that of many physicians, are of far less value. We shall mention these remedies and their indications in some detail, in order that we may refer to this account when the several forms of paralysis are under discussion.

When *electricity* is referred to as a means of cure of paralysis, the application of the faradic or of the galvanic current, which are now almost exclusively employed for medical purposes, is understood. As is well known, the use of the faradic current, as the chief curative means in the treatment of the most different kinds of paralysis, has been particularly recommended and practised by Duchenne, that of the galvanic current by Remak. There can be no doubt that both kinds of current are effective in the cure of paralysis, though their particular spheres of action have not as yet been detailed with sufficient accuracy. The galvanic current, however, incontestably possesses a much wider range of influence in paralysis than the faradic, especially because in many cases it constitutes the most effective means of attacking the causal indication. It is well known to be one of the most important remedial agents in many cerebral and spinal affections, accompanied by paralysis, in which the faradic current is almost inoperative; hence the pre-eminence which has been accorded to the galvanic current of late years in the treatment of paralysis. We are not called upon here, however, to speak of those actions of the electric currents that fulfil the causal indications (for which, as well as for the general mode of treatment of paralysis by electricity, we must refer to the chapters which are devoted to the galvanic treatment of diseases of the brain and spinal cord); we can here only discuss the proper "anti-paralytic" action of electricity and the modes in which it should be applied.

Great obscurity still exists in regard to the way in which the anti-paralytic action of faradic and galvanic currents is exerted. Most of the indications have been empirically discovered, and are not based on any precise knowledge in regard to the physiological action of electrical currents or of the nature of the processes producing paralysis. In by far the largest number of cases the *exciting influence* of electric currents is therapeutically employed. The fact, so surprising to some professional men, that muscular contractions and movements may be excited in the paralyzed parts by electrical irritation, long ago led to the opinion that it is just these artificially excited contractions which are so effective in the treatment. But just as no plausible explanation can be given of the kind and mode of this influence, so experience has not demonstrated that the occurrence of muscular contractions are unconditionally requisite for effecting the cure of paralysis. Nor can the result be referred to a simple re-establishment of conduction through the seat of lesion, since the current is, as is well known, usually applied below this point. In most cases it must be supposed that the electric or any other strong stimulus acting on the motor nerves has to overcome obstacles which in pathological cases are opposed to the conduction of the irritation, and hinder the transmission of stimuli to the muscles. If the obstacle which the will cannot overcome is subdued by violence, the path is then opened for the transmission of the impulses to the muscles, and the motility returns, though often at first the traces of it are very slight. In such cases the motility may often be observed to return quite suddenly after the application of a strong electric stimulus, and it is highly probable that by frequent repetitions of such strong motor irritation the path for conduction of the impulses of the will is permanently opened and a cure effected. This explanation is particularly applicable to those forms of paralysis which are accompanied by degeneration, trophic disturbance, atrophy of the nerves and muscles, and in which excitability and conductivity for strong stimuli first appear at a certain period of the regeneration. Frequent application of these strong stimuli restores the sensibility of the nerves and muscles for weaker stimuli. This object may be attained by the use of both kinds of currents, since these generally constitute a stimulus, and consequently induce muscular contraction. It is best and most certainly accomplished by the *direct method*, that is to say, by the direct application of the electric currents to the paralyzed nerves and muscles. Such excitations may however also be applied in an *indirect* or *reflex* manner. If the reflex arcs are situated on the central side of the paralyzing lesions, reflex stimuli analogous to those proceeding from the centre may penetrate into the paralyzed motor paths with each peripheric sensory stimulus, and gradually re-establish their conductivity by frequent repetition and progressive increase. In this reflex excitation, we have a valuable means of applying, in an indirect manner, the desired stimulus, which for anatomical reasons cannot be applied directly on the proximal side of the lesion. Those cases are of course most appropriate for the adoption of this method in which the simplest and commonest reflex connection is certainly preserved, as for example in cases of paralysis of the facial nerve, with intact trigeminus, or in those of the spinal paralysis of children where the sensibility of the paralyzed parts is always preserved. The conditions are naturally much more unfavorable in cases of peripheral paralysis of mixed nerves, in which the sensory nerves ministering to the reflex acts are coincidently paralyzed; but even here, in accordance with the laws of reflex irritation, excitations may be brought from remote sensory paths to paralyzed motor paths, though only by means of strong sensory stimuli. A moment's reflection shows that those paralyses, in which the reflex acts are preserved or increased, *cannot* be influenced by this means ; but that this is possible, on the contrary, in those in which the reflex acts are lost, provided, of course, that the reflex apparatus is not itself destroyed. It appears to me to be probable that many successful cases of peripheral electrical treatment, in central paralyses, are referable to those influences (though, no doubt, important results may perhaps be obtained by reflex action exerted upon the circulation at the point of lesion—results, however, which fulfil the causal indications). Both kinds of currents are likewise capable of exerting a reflex influence, though, of course, only when so applied as to produce strong sensory irritation.

There is some reason also for attributing a share in the beneficial effects of electricity in paralysis to its *power of increasing the excitability of the nerves.* It is a matter of fact that the transmission of either a faradic or a galvanic current through a nerve augments its excitability, and that the well-known electrotonic effects of the galvanic current, and especially the positive phase of the excitability after the action both of the cathode and of the anode, can be demonstrated in the nerves of the living man. It is alike possible and probable that frequent repetition of these actions may aid in the cure of many paralyses ; and this explains a part of the empirical successes.

Again, the *refreshing or reanimating effect* first observed by Heidenhain, which is produced where the galvanic current is transmitted through muscles exhausted by any previous stimulation, may also be regarded as a cause of its good effects in many forms of paralysis, especially when fatigue and exhaustion have resulted in the motor nerves or in the muscles from over-exertion. In such cases the ascending proves more serviceable than the descending current.

Lastly, we must not underestimate the *trophic influence* exerted by electric currents on the nerves and muscles, partly by causing muscular contractions, and partly by their action on the vessels and upon osmotic and chemical processes. No doubt too much stress has been laid on the therapeutic value of muscular contraction *per se*, since it has been imagined that the atrophy that occurs in so many cases might be arrested by the regular exercise of the muscles by means of electricity ; experience in traumatic and severe rheumatic paralyses has sufficiently demonstrated that this is not always the case, since in these cases the most persevering and timely employment of continuous and interrupted currents has no power to stay the progress of the degenerative atrophy in the muscles. On the other hand, the process of regeneration is unquestionably assisted by these procedures, and the instant regeneration commences in the nerves and muscles, methods of treatment should be adopted which improve the nutrition of the nerves and muscles by regular stimulation of the nerves and production of muscular contraction. By such means the return of the motility may be considerably assisted, and it is obvious that that current should be selected which is most effective in this direction, that in fact which must certainly liberate muscular contractions, influence the circulation, etc., and that is, in the vast majority of cases, the continuous or galvanic current.

It is at the same time quite conceivable that the actions of the electrical currents with which we are at present familiar are not those alone to which this great efficacy in the cure of paralysis is due, but that there may be others of which we are either wholly or in part ignorant. In the meantime, however, we must be contented to explain the phenomena observed by the light of our present physiological knowledge, and to refer to future research an exact and profound consideration of the causes of the anti-paralytic action of electric currents. The most advantageous methods of applying electrical currents in paralysis are the following:

Faradisation, the mode of action of which is tolerably simple. It consists in the first place, and in most instances, in directly or indirectly causing muscular contraction. Indirect irritation is much to be preferred, as a much feebler current is requisite, and its application is consequently less painful. It is sufficient to apply the ordinary rapidly interrupted induction current of the secondary coil; there is no advantage in applying the extra-current of the primary coil, with sometimes rapid, sometimes slow shocks, which was so strongly recommended by Duchenne. In central affections, diseases of the roots, etc., faradic currents, for physical reasons, rarely reach the true seat and focus of the malady in sufficient strength to be of service. We are in such cases limited to peripheral faradisation, and must chiefly rely upon its reflex action, and, if nothing else prove serviceable, the electric brush may be applied. Under ordinary circumstances, however, moist electrodes should be employed, and local faradisation systematically practised. The best technical rules and directions are to be found in the well-known treatise of Ziemssen. It is unnecessary to excite each muscle separately, but, by stimulating the nerve trunks implicated, the whole group of muscles may be excited to contract. As a general rule, the current should not be too strong; it is sufficient if muscular contractions are produced. In those cases in which faradic excitability is extinguished, the strength must be adapted to the sensibility of the skin, or to the effects produced on healthy parts. The sittings should be either daily or three times a week, and the duration of each from five to ten minutes, or, if the paralysis be very extensive, somewhat longer.

Galvanization.—We think it unnecessary to enter into particulars in regard to the galvanic treatment of the several causes of paralysis (cerebral, spinal, or peripheral disease), and beg to refer the reader to the several sections of this work devoted to those diseases. It may, however, here be mentioned that when galvanization is directly applied as an anti-paralytic agent, attention should always be paid to the seat of the disease, in order that the affected part, if possible, should be included in the circuit, as, for example, in diseases of the lumbar or cervical region of the spinal cord. It must ever be borne in mind that the treatment in loco morbi is that which has first to be attended to, and that the seat and nature of the disease must decide the choice of methods to be adopted. Without going into particulars, we shall here only say that in peripheral paralyses the application of the galvanic current at the place of lesion is indicated, whilst in spinal paralyses the current should be applied to the spinal column, and in cerebral paralyses to the head and sympathetic nerve, though, of course, with all the precautions and attention to the contra-indications mentioned in the special sections. For the really anti-paralytic effects the cathode, applied both in a stabile and a labile manner, is specially indicated as fulfilling almost all the indications. It constitutes a stimulus, and at the same time it increases the excitability of the nerves ; it leaves behind it positive changes in them, and by its means muscular contractions are most easily produced. It should be brought everywhere as freely as possible into contact with the paralyzed parts—nerves and muscles,—which may best be accomplished by stroking the skin with the cathode in the direction of the nerves and muscles to be acted on (so-called labile application). The anode is most advantageously placed over the seat of the paralysis, or upon the plexus, or upon any indifferent part of the body ; it may also be placed, in order to obtain the refreshing action of the ascending current, on the peripheral side of the cathode. The principal influence, however, is always exerted through the labile application of the cathode. Stimulation may also be caused by the interruption of the current, which may best be accomplished by repeated cathodal closures, or, in a still higher degree, by reversals of the current, the so-called voltaic alternatives. These measures may be resorted to when a stronger irritation is desired, or when there is a very low grade of excitability, as for example in the later stages of the reaction of degeneration. The strength of current selected should be such that distinct contractions, moderate sensation of burning, and redness of the skin should be produced. The sittings should last for four, six, or ten minutes, and be repeated every day, or every two or three days, according to the nature and seat of the disease. the peculiarities of the case, and other circumstances.

It is seldom that any instantaneous or strikingly rapid result proceeds from faradisation or galvanization ; cases, however, do now and then occur in which, after a single sitting, sudden restoration of motility is observed in parts which have remained incurable for weeks, months, or years (as, for example, in paralyses of the chordæ vocales, in traumatic paralyses which are considerably advanced towards regeneration, in slight rheumatic paralyses when they have existed for some time, and in some cerebral and hysterical paralyses). As a rule, more or less time elapses before motility is restored, and a still further period is required before it returns completely to its normal condition. We must not, however, be misled by the treatment being apparently unsuccessful for a long time ; patience and perseverance are indispensable requisites in the electric treatment of paralyses.

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It is in paralyses with the reaction of degeneration that our patience is especially subjected to severe trial, since it appears as if the electric treatment was entirely without influence in causing the reappearance of the motility, though it undoubtedly hastens its restoration when it has once commenced. In regard to all these points, there are so many special and accidental conditions to consider that we must here refer to the sections upon the several forms of paralyses and to the special treatises on electro-therapeutics.

We shall not here enter into the question, which has so often been warmly discussed, as to whether the galvanic or the faradic current is the most effective in the cure of paralysis. This much, however, is certain, that neither of the two kinds of currents is invariably successful. Still no one will now be found to deny that the galvanic current has a much greater range of influence than the faradic, because it proves serviceable in many central paralyses in which the faradic current is quite inoperative. On the other hand, experience shows that the faradic current has yielded many favorable results, and it may therefore be employed in many cases. It sometimes even appears that the alternate application of the two currents has a particularly beneficial effect. In those cases in which the superiority of the galvanic current has been enthusiastically claimed, as in severe rheumatic facial paralyses, and traumatic paralyses with the reaction of degeneration, critical and unbiased observation has shown without a doubt that neither the faradic nor the galvanic current exercises any decided influence upon their course or at least upon the rapidity of their course. We have already mentioned that in such cases the progress of the muscular atrophy cannot be arrested by any kind of electrical treatment. The exclusive employment of the galvanic current, as a means of cure in paralysis, has been recommended by very many authors; yet this recommendation does not appear to rest upon a sufficiently secure basis. The chief ground for the employment of the galvanic current lies in the catalytic effect which it exerts at the point of lesion, in the fact that it hastens the commencing process of recovery by its powerful action upon the vessels and upon the trophic processes taking place in the regenerating nerves and muscles; and this is certainly in some measure justified by clinical experience.

In regard to certain contra-indications to the electric treatment of paralyses, we must refer to the sections of this work devoted to the several forms of paralysis and to the various treatises on electro-therapeutics.

Different kinds of *baths* have, from the most ancient times, been highly celebrated as means of cure in paralysis, although they do not admit of such varied application as electricity. It is very difficult to distinguish the direct anti-paralytic action of baths from those which satisfy the causal indications. The "enlivening" and "tonic" action claimed for various spas tells us very little, since this affords no explanation of their anti-paralytic action. It is extremely probable that the principal influence exerted by baths consists in the removal of such causes of paralyses as exudations, inflammatory processes, extravasations, degenerations, etc. The removal of these conditions effects the cure of the paralysis, but the mode in which this is accomplished by baths, has not, as yet, been sufficiently explained, and we must refer on this point to the treatises on balneo-therapeutics. A direct anti-paralytic influence, due to the excitations of the motor apparatus, can scarcely be ascribed to any form of bath, whether the saline baths, the gaseous saline baths, especially those impregnated with carbonic acid, sea baths, pine-needle baths, cold-water cure, etc., but we most rather refer their action to indirect reflex excitation proceeding from the skin. The mechanism of these actions is, however, extremely obscure; nevertheless the trials of them and the empirical successes that have been obtained are sufficiently numerous and brilliant to give baths a prominent position in the treatment of paralysis.

The *indifferent thermal baths*, as those of Schlangenbad, Wildbad, Pfäffers, Ragatz, Gastein, Teplitz, Wiesbaden, Warmbrunn, Leuk, Plombières, etc., have always had a great renown in the treatment of paralyses, and fulfil very different indications, according to their temperatures and elevations above the level of the sea; very recently, also, the highly gaseous, thermal saline baths of Rehme and Nauheim have been highly praised. In addition, cold saline baths sea baths, cold-water cures, sulphur baths, pine-needle baths, vapor baths, hot-sand baths, animal baths, have been found serviceable in many paralyses; but as we have at present no exact knowledge of their mode of action, and are consequently unable to give any precise indications for their use, we shall here limit ourselves to a short *résumé* of the more important principles that have been established by experience, referring for all details to the special treatises.[1]

Paralyses resulting from exudation processes, spinal and cerebro-spinal meningitis, simple chronic myelitis, rheumatic affections, blood extravasations and the like, are those that receive most benefit from the indifferent thermal baths, or the thermal saline baths. Tabetic paralyses are less appropriately treated by thermal baths, but are far more benefited by thermal saline baths or moderately cold-water cures. The very hot baths of Teplitz, Gastein and Wiesbaden are best adapted for the treatment of chronic rheumatic forms, though these may also be treated successfully by the energetic application of the cold-water cure. Moderately warm baths, as those of Schlangenbad, Ragatz, or at most Wildbad, are alone applicable to hemiplegiæ caused by apoplexy. In these affections, elevated watering-places with saline baths, or light cold-water cures, frequently prove more beneficial; in paralyses from lead, sulphur baths are held in great esteem.

In paralyses proceeding from exhaustion, losses of blood, serious disease, etc., climatic cures, mountain or sea air, combined with indifferent or saline thermal baths, pine-needle baths, etc., are indicated. For these cases the cold-water cure is usually too exhausting. A combination of a course of thermal waters with galvanic treatment appears to be particularly useful in many cases of severe traumatic (gunshot wounds) or rheumatic exudative paralysis, and this combination has been also recommended in many other forms of paralysis (Karmin). Improved diagnosis and a more accurate knowledge of the effects of baths will enable us to give better indications for their use.

The *gymnastic cure*, or *Swedish gymnastics*—as it may be termed in opposition to ordinary hygienic gymnastics—is of subordinate importance in the treatment of paralysis. It aims, by a methodic exercise of the paralyzed muscles, aided by some skill and expert guidance, to produce more active voluntary contractions. The nutrition of the muscles is thereby improved and their excitability and dependence on the will increased. It is obvious that when the paralysis is complete, no attempts to effect the gymnastic cure can be made, because no voluntary movements can be executed. On the other hand, in pareses and in paralyses where recovery has commenced, gymnastics may advantageously act in hastening the progress of the cure. It is easily intelligible, however, that the range of action of the gymnastic cure in paralysis is very limited ; in fact, it proves chiefly successful in other kinds of disease, as in contractures and in orthopædic surgery.

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[1] See Braun, Balneotherapie, 3. Aufl., p. 569, et seq.

In many forms of paralysis the motility may be improved by simple active movements. With this object in view, systematic and frequently repeated movements, either of a simple or complicated nature, should be executed with the paralyzed parts, as, for example, the various kinds of in-door gymnastics, exercise with tools, methodic raising of weights, etc.

The effect of such active movements may be augmented by the opposition of a practised assistant, who should exert himself in accordance with the particular circumstances of the case, his efforts constituting the so-called duplicate, or resisting movements of the Swedish school. If, for example, the extensors of a joint are to be exercised, the limb must be brought into a flexed position, and the patient required to extend it, whilst the assistant opposes the extension with more or less force ; such exercises can, however, only be practised where the muscles are already tolerably vigorous. In other cases, in order that a desired movement should be effected, it is necessary to aid the contraction of the muscles, and to overcome the opposition of the antagonists. It can easily be shown that in every voluntary innervation of a given group of muscles, the antagonistic muscles, and even those at some distance, are coincidently excited to action to a greater or less extent ; and this is done in order to fix the position of the joint, to insure uniformity in the movements, and to preserve the equipoise of the body. But if a group of muscles be paralyzed, any attempt to innervate them excites the involuntary but physiologically induced contraction of their antagonists, a contraction which cannot be suppressed, and thus movement of the paralyzed muscles is rendered impossible. If this action of the antagonists be eliminated by passive resistance, which at the same time aids the desired contraction of the enfeebled muscles, even weak and slight contractions of the latter may be induced, and then gradually promoted by repeated exercise. The amount of this contraction may be increased, and the nutrition thereby gradually improved by passive approximation of the attachments of the paralyzed muscles. The most diverse paretic groups of muscles may have their activity assisted, and their motility gradually augmented by systematic exercise, aided by the hand or by a practised assistant, or by appropriate elastic bands and machines.

The passive movements just mentioned are particularly applicable to those cases where mechanical obstacles, which are opposed to the contraction of the paralyzed muscles, have to be overcome, the removal of which is necessary before a cure of the paralysis is possible. Contractures, stiffness, and anchylosis of the joints are cases of this kind, and in these much may be effected by patience and perseverance.

The mode in which these gymnastic proceedings exert an influence, consists, no doubt, in occasioning frequently repeated voluntary excitation of the nerves and muscles, so that the act of conduction to the muscles is gradually rendered more facile, and ultimately the nutrition of the nerves and muscles is augmented. The most favorable results are seen in those pareses which long remain after paralyses, whether of peripheral or central origin. The progress of recovery in these cases is often essentially facilitated by systematic gymnastic exercise. Good results may also be occasionally seen to follow the adoption of the same measures in hysterical paralysis, if we are successful in exciting the patient to make an energetic exertion of the will. The cases in which benefit is chiefly derived from gymnastics, are, however, those where deformities follow paralysis; but the treatment of these affections belongs to the domain of orthopædic surgery.

In all cases the method adopted should be based on a precise diagnosis and on reasonable physiological grounds, and when once commenced should be pursued with care for a considerable period. We may discuss in a few words the consideration of those *internal remedies* that are not exclusively directed to the removal of the cause of the disease, but which are claimed to have a direct anti-paralytic action. Their number is very small, and the benefit to be obtained from them, in many cases at least, very doubtful. The preparations of nux vomica, and particularly of strychnia and brucia, are very commonly prescribed in paralyses of cerebral and spinal origin. At present it is only known that strychnia increases the reflex excitability of the spinal cord, and, when taken in large doses, acts as an irritant to it, whilst it does not seem to have any direct influence upon the brain or motor nerves; it is therefore not quite clear *how* it acts, though experience undoubtedly shows that it is really serviceable in many forms of paralysis. It may be employed in the more chronic cases, those which have become stationary and in which no phenomena of irritation are present. As soon as the smallest indications of its toxic influence appear (psychical disturbance, restlessness, stiffness, and tension of the muscles, spasms, especially in the paralyzed parts, exhaustion, etc.), its use should be discontinued. The extract of nux vomica is given internally in quantities of from one-sixth to three-quarters of a grain, and nitrate of strychnia in doses of from one twenty-second to one-sixth of a grain; the latter alkaloid may be administered by subcutaneous injection, commencing with one-thirtieth of a grain. The best results appear to have been obtained in cases of vesical and rectal paralysis, especially when of spinal origin. It has, however, been recommended and used in a great variety of central, as well as of peripheral, rheumatic, and similar paralyses. The benefit derived is often very striking (Hunter). Numerous cases have been recorded, but none of them afford any satisfactory clue to the special indications for its use. Still less can be said of the other internal remedies formerly so much employed. Arnica does not justify its reputation as a nerve invigorator. Ergot appears to act chiefly on the unstriped muscular fibres, and has become celebrated for its utility in weakness of the bladder. Cantharides and turpentine have properly fallen into complete disuse, and arsenic should be prescribed only under very peculiar circumstances.

For a long time past certain external remedies have been employed in the treatment of paralysis, and are much esteemed by the profession and by the laity. Such are embrocations of all kinds, cutaneous excitants, frictions, actual cautery, douches, etc. Their action, however, is only very subordinate and unimportant ; still some may exercise a favorable influence on paralyses by exciting reflex actions, or by invigorating the circulatory and secretory organs, or even by the simple mechanical action of rubbing and shampooing. Many of these means are adapted to fulfil the causal indication by their action as derivatives, and this affords sufficient explanation why they have become so firmly established in practice ; to such applications belong stimulating embrocations, etc.; such as the liniment of camphor, oil of turpentine, spirit of ants, spirit of wild thyme, ointment of veratria, ointment of cantharides, etc. ; also *local baths* of warm water, with salt, malt, and animal baths, local douches, especially with cold and hot water alternately ; lastly, strong derivatives, flying blisters, moxæ, the actual cautery, issues, etc. These several remedies have only been enumerated in order that the advice may be added, that too much time should not be lost in employing them, and that too much reliance should not be placed upon them. It may be observed, however, that Weir Mitchell[1] found a combination of electricity and shampooing, with alternate cold and warm douches, extremely effective in traumatic paralyses. From the preceding accounts of the remedies in ordinary use against paralysis, the *general plan of treatment* to be adopted for the cure of this disease may easily be deduced. The first and most important is the determination and fulfilment of the indicatio causalis. Where direct anti-paralytic treatment is requisite or adopted from the commencement, electricity should first be used. Providing there is no special contra-indication, the proper choice and alternation of the other remedies must depend upon the tact of the physician and the special circumstances of the case. A judicious economy and due alternation in the choice of remedies is very permissible, considering the long duration and the slow progress of recovery, in order to allay the impatience of the patient.

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[1] Loc. cit. p. 250, where a precise account is given.

No definite rules can be laid down for the hygienic and dietetic treatment that should be adopted in the different forms of paralyses. It must in almost all instances be determined by the nature of the primary disease. A few words may, however, be added in regard to the care to be taken of the paralyzed parts. They should be carefully protected against injury, especially in those cases where accidents are likely to occur in consequence of the presence of marked anæsthesia combined with vaso-motor and trophic disturbances. Bed-sores must be guarded against by appropriate position, washing, and bandaging. Lastly, we may briefly observe that careful attention and timely treatment should be given to disturbances of the excretion of urine, because dangerous affections frequently arise from them.

4. Particular Forms of Paralysis.

a. *Paralyses of the Muscles supplied by the Oculo-motorius, the Trochlearis, and the Abducens. Paralyses of the Muscles of the Eye.*

Ch. Bell, Physiol. u. pathol. Unters. Deutsch v. Romberg, 1832.—*Romberg*, Nervenkrankh. I. 3. *pp.* 63, 77. 1851.—*V. Graefe*, Symptomenlehre der Augenmuskellähmungen. Berlin, 1869.—*A. Eulenburg*. l. c. p. 469.—*Benedict*, Arch. f. Ophthalm. X. 1. 1864. Electrother. 1868.—*M. Rosenthal*, Electrother. 2. Aufl. Lehrb. d. Nervenkrankheiten.—*Struthers*, Paral. of the common motor ocul. nerve. Monthl. Journ. of med. Sc. 1853.—*Brenner*, Petersburgh med. Ztschr., XII., Heft 5. 1867.—*Gozzini*, L'elettroter. nella paralisi degli oculomotori. Gazz. med. it. Lomb. 1868.—*W. Erb*, z. galv. Behand. von Augen- und Ohrenleiden. Arch. f. Augen- und Ohrenheilk. v. Knapp u. Moos. II. 1. 1871.—*Driver*, Behand. einiger Augenleiden mit d. const. Strom. ibid. II. 2. 1873.—Consult also the text-books on Diseases of the Eye by *Arlt*, *Stellwag*, *Schweigger*, and others.

The great importance of paralyses of the muscles of the eye in neuro-pathology and the frequency of their occurrence, either alone or as concomitant symptoms of different cerebral and spinal diseases, oblige us to give a brief account of their pathogenesis, symptomatology, and treatment. For the details of their special diagnosis we must refer to works devoted to ophthalmology. We shall discuss these forms of disease collectively, since they present many points of similarity, and can only be distinguished from one another by their symptoms.

Etiology.—Paralyses of the muscles of the eye are of very frequent occurrence, and arise from very different causes. *Catching cold* is a very frequent cause (rheumatic paralysis of the ocular muscles), and seems to affect chiefly the abducens and the oculo-motor nerves, of which occasionally only a few branches are attacked; the trochlearis is seldom affected. Our knowledge of the real nature of the rheumatic affections of the nerves supplying the muscles of the eye is as imperfect as that of other rheumatic paralyses, and we cannot determine with any degree of certainty whether its seat is in the orbit or at the basis cranii, as the symptomatological data are too uncertain. Struthers found in one case of paralysis of the oculo-motorius, of several months' duration, and probably of rheumatic origin, atrophy and sclerosis of the nerves at the base of the skull.

Wounds affecting the tissues in the orbit or cranium constitute the next most common and important cause; thus paralyses of the muscles of the eye have frequently been seen to arise from blows on the eyes, from penetrating wounds made with a sharp weapon as a knife or needle, from a blow given by the horn of an animal, or from fractures of the skull. Mechanical compression of the nerves, however produced, has a similar effect, and the causes leading to the compression may either be situated in the orbit, such as tumors, aneurisms, extravasations of blood, periostitis, etc., or at the base of the cranium, tumors, meningitis, aneurisms, syphilitic affections, etc. Leber noticed atrophy and flattening of both nervi abducentes, where they accompany the carotid artery, in a case of augmented intra-cranial pressure from cerebral tumor. The cases which originate in neuritis may also be included amongst the foregoing, and I have seen several that resulted from erysipelas which had extended into the orbit, and others from acute meningitis.

Diseases of the brain are very frequently accompanied by paralyses of the ocular muscles. This occurs, for example, in apoplexy, in tumors, and other local affections, especially in the pons and cerebral peduncles, in the vicinity of the central ganglia, and near the fourth ventricle. It also occurs in many forms of bulbar paralysis and progressive paralysis of the cerebral nerves.

Affections of the spinal cord, especially tabes dorsalis, and allied forms of disease, are not unfrequently accompanied by paralyses of the muscles of the eye. Pareses of the oculo-motorius and abducens occur in the early stages of tabes, and are not unfrequent in its later stage; they possess a high diagnostic value, and depend for the most part on anatomical changes, which have extended from the spinal cord to the corresponding nerve centres and the nerves themselves.

A cause which may act in different ways, and which, on account of its frequency, is deserving of separate mention, is *syphilis*. Paralyses of the ocular muscles are, indeed, amongst the commonest nervous symptoms that occur in the later stages of syphilis, and may be occasioned by very various anatomical changes, as by periostitis and exostoses in the orbit or at the base of the skull, or by gummata at various points in the course of the nerves or in the brain.

Diphtheritis must also be mentioned, since it is a relatively frequent cause of paralysis of certain branches of the oculo-motorius (paralysis of accommodation, mydriasis). Lastly, ocular paralysis is not unfrequently observed after acute diseases. Strong impressions of light, excessive smoking, the abuse of alcohol, and similar irregularities are known to constitute causes of the affection.

Symptomatology.—The symptoms of ocular paralysis have, under von Graefe's intelligent direction, been investigated with extraordinary thoroughness, and the corresponding lesions determined with great precision by the aid of relatively simple means; this depends upon the extreme exactness with which the associated and accommodative action of the ocular muscles

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takes place in binocular vision—which indeed, without such exactness would be impossible. The slightest disturbance in the function of either of the ocular muscles immediately betrays itself by disturbance of the vision—by *diplopia* and unsymmetrical position of the globes—the so-called *strabismus paralyticus.* By skilful investigation and determination of the double images (for which purpose colored glasses and prisms are employed), by exact testing practised with a view of determining the monocular and binocular fixation of the eye, by observation of the mode in which the head is held, etc., the affected muscle may be very exactly determined and the degree of its functional disturbance ascertained. For further particulars we must refer to von Graefe's works, from which we make the following brief abstract.

Paralyses of the muscles of the eye betray themselves, first, by some *defect of the absolute mobility* of the eye in the direction of the action of the paralyzed muscle. This defect is only striking when the paralysis is complete, and in simple paresis the symptoms may be very slight, since, on account of the easy movement of the globe, the power required for its rotation is extremely small. As a rule, the defect is only perceptible when the two eyes are compared, by which means alone can it be ascertained with certainty whether the deficient movement is, or is not, within the limits of health. An *abnormal mode of movement* is sometimes associated with the defect, and is indicated by a spasmodic or jerking contraction of the paralyzed muscles, or by concomitant actions of other muscles leading to abnormal rotation of the globe, etc.

The recognition of paralysis of the muscles of the eye is rendered more certain by the defect *of its relative mobility*, as compared with the associated movements of the other eye. Since, under normal conditions, a uniform impulse of the will acts, always, on the coincidently active muscles of both sides, that muscle, which is suffering from diminished functional activity (paretic or paralytic), lags behind the opposite healthy one in its contraction; the extent of rotation of the eye is more limited, and that limitation becomes objectively visible as a squint or strabismus, which makes its appearance whenever the contraction of the weakened muscles is required for binocular vision. The immediate functional result of this relatively smaller rotation of the eye on the affected side is *double vision—diplopia*—which is the pathognomonic symptom of all paralyses of the ocular muscles.

This lagging behind of the globe, and consequent separation of the double images, augments in proportion as the object looked at approximates that part of the field of vision which is commanded by the paralyzed muscle; and it is just this *progressive increase of the deviation and of the diplopia* towards the side of the paralyzed muscle which distinguishes it from concomitant strabismus. In order to make use of diplopia, as a means of ascertaining which muscle is affected, von Graefe has given a very simple rule, namely: that if the double images occur on any side of the common field of vision (right, left, upper or lower), that eye is affected with paralysis in which the image appears most advanced towards the same side, this eye having more or less lost the power of being rotated in that direction.

For example, if the double images occur on the left side, then if the image of the left eye lies towards the left, (homonymous diplopia), this eye is the affected one, and the muscle which should turn it in this direction, the abducens, is that which is paralyzed. If with similar position of the object, the image of the right eye is directed more to the left (crossed diplopia), the right eye is affected, and the force which should rotate the eye to the left, that is to say, the rectus internus, is paralyzed. The same rule holds good, mutatis mutandis, for the right upper and lower segments of the field of vision.

Homonymous and crossed double images thus occur, for which the rule holds good that, in pathological *convergence* of the visual axis, *homonymous*, in pathological *divergence*, *decussated double images* are perceived.

In many cases a so-called overlapping or confused *double vision* may be observed, in which the patient only complains of indistinct vision or is only incommoded in certain parts of the field of vision; this condition is characterized by the vision being perfectly good with each eye alone. The double images can usually be rendered very apparent by the use of colored glasses with direction of the attention to them.

The secondary deviation of the sound eye is very characteristic, and constitutes a valuable aid to the diagnosis of slight pareses. It occurs when the sound eye is covered with the hand, and the affected eye is chiefly or exclusively used for fixing the object. If the fixed object be moved in the direction of the action of the paralyzed muscle, this muscle will have to make progressively increasing efforts to preserve the fixation. The same efforts will, however, in accordance with the law of association, be made by the muscles of the sound side, and the healthy eye is thus thrown into a state of deviation much greater in degree than that of the affected eye when the sound one is used for fixation. This is owing to the circumstance that, in the former case, much stronger nervous efforts are made to which the healthy eye responds. Thus, for example, in paralysis of the rectus externus, if some object is held to the outer side of the affected eye, a strong secondary deviation, inwards, is produced in the sound eye by the action of the associated rectus internus, and so correspondingly in paralysis of every other muscle. It is characteristic of this secondary deviation that it is greater than the primary deviation of the paralyzed eye (always oppositely directed), and that it increases in proportion to the resistance to the innervation of the affected muscle ; it thus forms to some extent a measure of the amount of this disturbance of innervation. The fact that it is greater than the primary deviation is of great value in the diagnosis of very slight and scarcely perceptible disturbances of the affected eye, particularly in those muscles that exhibit when paralyzed so small a deviation as the obliquus superior. The greater amount of the secondary deviation may here greatly facilitate the diagnosis.

A symptom that is very annoying to the patient is the *false projection of the field of vision.* In consequence of the increased muscular effort that is made with the affected eye, the amount of rotation effected is over-estimated, and consequently an erroneous judgment is formed of external objects. The field of vision is displaced in the direction of the action of the paralyzed muscle : in paralysis of the abducens, for example, towards the outer side and in paralysis of the rectus inferior, downwards. If, in paralysis of the abducens, the patient grasps quickly at an object held before him in an appropriate direction, he will miss it by going too far to the outer side.

This symptom is particularly noticeable when several muscles are coincidently affected, and when the patient is obliged, as in certain occupations, frequently to change the field of vision alternately in opposite directions, as in walking. The false positions of the field of vision and the apparent movements of the objects produce a feeling of great insecurity, and the *giddiness* which is so frequent and characteristic a symptom of paralysis of the ocular muscles. This occurs especially when the paralyzed eye is alone used, and is not to be confounded with the unpleasant feeling which the perception of double images occasions in binocular vision ; the latter sensation may sometimes rise to actual pain, caused by the abnormal efforts at contraction that are made to avoid the double vision. The pain occurs, therefore, principally when objects are looked at in certain directions.

Some of these troublesome symptoms may be avoided by the patient: first, by *covering the affected eye*, so as to prevent its use and to escape the unpleasant sensation of double images ; and, secondly, by *holding the head in a peculiar position*, so that that part of the field of vision only is used in which no double images occur. These positions of the head are very characteristic, since they differ in accordance with the different muscles that are paralyzed. The head is then generally kept rigidly in the same position.

A similar advantage can be obtained in certain minor pareses, particularly of the muscles effecting horizontal rotation, by the *possibility of effecting fusion of double images.* This only exists to a certain small extent, though varying in particular cases, but may entirely conceal the existence of diplopia. This may, however, generally be demonstrated by examination with proper prismatic glasses refracting upwards or downwards. So, too, by examination with proper prisms, the power that exists of combining more or less widely-separated double images may be ascertained. This may prove of importance in the diagnosis of the seat of the paralysis.

Of scarcely less importance is, lastly, the secondary contracture of the antagonistic muscles, so common in many paralyses of the eye. This occurs in different cases in very different degrees of intensity, partly owing to the unilateral function of these muscles, partly to the loss of the opposition formerly offered by the paralyzed muscle, and the consequent gradual trophic shortening, and partly also, in many instances, to active irritation. It is obvious that this condition increases the extent of deviation, augments the distance of the double images from one another, and extends the limits of diplopia more and more over the region of the originally paralyzed muscle towards the opposite side. The diplopia caused by the contracture remains, however, about the same on the side of the contracted muscle, whilst it progressively increases on the side of the paralyzed muscle.

After these general observations, the special symptoms of the different paralyses of the ocular muscles may be rather briefly given.

1. *Paralysis of the oculo-motorius.*—This nerve innervates the levator palpebræ superioris, the rectus superior, the rectus internus, the rectus inferior, the obliquus inferior, the musculus sphincter iridis, and the musculus accommodatorius.

Complete paralysis of the oculo-motorius, and, consequently, of the above-named muscles, presents a very characteristic appearance. The upper eyelid hangs motionless, reaching to the lower border of the cornea, and cannot be raised (ptosis). The eye is almost completely motionless, yet when at rest is still tolerably straight, providing no secondary contractures have taken place in the unaffected muscles (rectus externus and obliquus superior). The movements inwards, upwards, and directly downwards are completely abolished; on the other hand, rotation outwards is preserved. No movements can be made either upwards and outwards, upwards and inwards, or downwards and inwards, but the eye can still be rotated downwards and outwards. Every effort to move the eye causes it to rotate downwards and outwards, owing to the action of the external rectus and superior oblique, and it gradually becomes fixed in this position by the contracture of these muscles. The pupil is dilated and immovable, and the power of accommodation is greatly diminished. Fixation is only possible in a downward and outward direction; double images appear in almost the whole field of vision. Secondary deviation of the healthy eye takes place in all directions, with the exception of that towards the affected eye. The eye is frequently slightly protruded, producing the so-called exophthalmus paralyticus, because almost all the straight muscles of the eye are paralyzed. The acuteness of vision may, however, be perfectly normal. False projection of the field of vision occurs in every direction towards which futile efforts of fixation are made. Hence, the feeling of giddiness is very strongly marked and annoying, if, indeed, vision be not altogether prevented by the ptosis. The eye is generally kept closed intentionally by the patient. The position of the head is also a very oblique one, being turned backwards and towards the healthy side, if this be not rendered superfluous by the occurrence of ptosis.

Partial paralysis of the oculo-motorius may either affect a single muscle or several muscles.

Paralysis of the levator palpebræ superioris—Ptosis.—This form of paralysis may be quite isolated, as when it arises from injuries, or even spontaneously; it is frequently associated with paralyses of the superior rectus, which is also supplied by the superior branch of the oculo-motor nerve; the upper lid hangs motionless; the fissure of the lids is greatly narrowed; and even when the eye is directed upwards, the lid is not raised; it is only very slightly elevated when the healthy eye is closed and the other is directed upwards; the auxiliary contraction and influence of the frontal muscle is very characteristic, causing elevation of the eyebrows and deep wrinkles across the forehead. As improvement takes place, the lid gradually rises higher and higher.

Paralysis of the rectus superior.—The power of turning the eye upwards is impaired, so that the line of vision is either not at all or but slightly raised above the horizontal plane; deviation downwards occurs, the visual axis being directed more downwards than in the sound eye—strabismus deorsum vergens; the cornea diverges a little outwards from the action of the obliquus inferior. The double images are superimposed vertically and slightly decussate, that of the paralyzed eye being the higher of the two, and the vertical distance between them increasing with the elevation of the point of fixation. They disappear at the horizontal line (providing there is no contracture of the inferior rectus). Objects in the lower half of the field of vision are seen single. Secondary deviation of the healthy eye takes place upwards and somewhat outwards. The field of vision of the affected eye is projected upwards, and the head is depressed in order to avoid the double images. A feeling of giddiness rarely occurs, and indeed is only felt on climbing a steep staircase or a ladder. When secondary antagonistic shortening takes place, the double images extend towards the lower part of the field of vision, though only to a less extent. This paralysis may also be isolated ; it is, however, frequently associated with ptosis.

In *paralysis of the rectus internus* there is impairment of the power of rotating the globe inwards, and, when the paralysis is complete, no movement can be effected beyond the vertical line ; there is deviation of the eye outwards, and the axis of the eye is inclined outwards—strabismus divergens. The double images are side by side and crossed, the image of the diseased eye being on the healthy side. The lateral distance between the images increases with the movement of the object towards the sound side ; objects appear single in the outer half of the field of vision, providing no antagonistic contractures be present. There is secondary deviation of the sound eye outwards. The field of vision of the affected eye is projected inwards, the head is turned towards the healthy side. When antagonistic contracture has taken place, the diplopia extends into the outer half of the field of vision ; the distance of the double images becomes greater, but remains stationary externally, and increases progressively towards the inner side.

Paralysis of the rectus inferior.—The symptoms are here exactly the inverse of those observed in paralysis of the rectus superior. The mobility of the eye downwards is impaired, the globe remains at a higher level, the visual axis deviates upwards—strabismus sursum vergens ; the cornea is also turned a little

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outward, owing to the action of the obliquns superior; the double images are vertical, slightly oblique, and crossed, the image of the affected eye being lowest. Objects appear single in the upper part of the field of vision. There is secondary deviation of the sound eye downwards and somewhat outwards; the head is inclined forwards; the feeling of giddiness is very marked. This form of paralysis is particularly troublesome whenever the line of vision is lowered, as in walking, in all kinds of handiwork, climbing stairs, etc. When secondary contracture of the rectus superior occurs, the corresponding symptoms are produced.

Paralysis of the obliquus inferior.—The defective mobility of the eye is not here very readily perceptible, but becomes most obvious in the upper and inner angle of the field of vision; it is often more easily recognized by the secondary deviation upwards and somewhat outwards of the healthy eye. Deviation of the affected eye occurs to some extent downwards and inwards; double images are chiefly observed in the upper half of the field of vision, standing not quite vertically to one another, and being homonymous and somewhat oblique. Their vertical distance increases as the object is moved inwards, and their inclination to one another augments with rotation outwards. There is a false projection of the field of vision upwards, or somewhat outwards and upwards, on fixation on the median plane; the head is thrown backwards and the chin turned a little towards the healthy side, so that the lower and outward segment of the field of vision is chiefly brought into use. When secondary shortening of the obliquus superior has taken place, the double images extend into the lower half of the field of vision.

If several of these muscles, as not unfrequently occurs, are coincidently paralyzed, the symptoms are combined and may become very complicated and difficult to decipher. The details of the diagnosis cannot here be considered.

Paralysis of the sphincter iridis.—Mydriasis paralytica.—In this affection the pupil is in a medium state of dilatation, and is nearly or completely motionless when subjected to the stimulus of light; it contracts also very little, or not at all, when the eyes are strongly converged, or when efforts of accommodation are made; it may be still further dilated by atropine. Vision is rendered indistinct, in consequence of circles of dispersion; bright light is intensely disagreeable; the power of accommodation is often diminished, but may be completely preserved.

Paralysis of the musculus accommodatorius (ciliary muscle).—This affection may occur quite independently, but is often also complicated with mydriasis; in this form of paralysis the focal distance is increased; the patient finds himself incapable of focusing near objects, or of reading small print; he hence often forms an incorrect estimate of the size and distance of objects. Paralysis of accommodation is one of the most common symptoms of diphtheritic paralysis; it may also accompany any other paralysis of the third nerve.

2. *Paralysis of the trochlearis nerve.*—One muscle alone, the superior oblique, is here rendered functionally inactive. This paralysis is not easily recognized, because the fourth nerve is almost always associated in its action with other muscles, and its function can be in part performed vicariously. There is usually only very slight defect of the motility of the eye; what there is, occurs chiefly in the inner and lower angle of the field of vision; there is deviation of the eye inwards and upwards on lowering the object, and simply upwards when it is turned far towards the healthy side. Double images appear in the lower half of the field of vision, on the inner side (rising somewhat above the horizontal level, and on the outer side falling somewhat below it), vertically superimposed, homonymous, and somewhat oblique, their distance increasing towards the healthy side. The secondary deviation is usually directed straight downwards, and is very well marked; there is false projection of the field of vision, downwards and a little outwards; the feeling of giddiness is often very well marked; the head is inclined forwards, and turned towards the healthy side (the affected eye being rotated towards the chin), so that objects are brought into the upper and outer quadrant of the field of vision.

When antagonistic shortening of the inferior oblique exists, the deviation and diplopia extend more and more into the upper half of the field of vision. Paralysis of the trochlearis often occurs as an isolated affection in syphilis, from injury, and rarely from exposure to cold.

3. *Paralysis of the nervus abducens.*—In this case also one muscle alone, the rectus externus, is paralyzed; the symptoms are not complex. There is defect of the absolute mobility of the eye outwards, so that the globe cannot be rotated outwards beyond the middle line, or, if at all, only by a great effort, which is accompanied with spasmodic tremulous movements, and even this is impossible when secondary contracture has taken place. There is deviation of the eye inwards, or strabismus convergens; the patient experiences diplopia in the external half of the field of vision; the double images are lateral and homonymous, and their distance increases as the object is moved outwards. Secondary deviation occurs towards the inner side, and is very well marked; the head is turned towards the affected side; there is false projection of the field of vision towards the outer side; the feeling of giddiness is moderate, and is especially experienced when the affected eye alone is employed during rapid movements. When secondary shortening occurs, the double images extend inwards, and often affect the whole field of vision.

Paralysis of the sixth nerve is very often an isolated affection, especially when due to rheumatism. It is the commonest rheumatic paralysis of the ocular muscles.

It is sometimes bilateral, as in tabes dorsalis, or as occurs after acute cerebral meningitis.

The course of the different paralyses of the ocular muscles varies exceedingly, according to the pathological condition to which they owe their origin.

The paralysis sometimes supervenes quite suddenly, appearing in the course of a night, as, for example, in the rheumatic and apoplectic forms. In other instances it develops slowly and gradually, and with considerable fluctuations, as when associated with syphilis, neuritis, and chronic disease of the central nervous system. The patient experiences, in the first instance, a certain amount of discomfort in looking at objects, especially in certain directions, and this gradually or suddenly manifests itself as distinct double vision. The degree of interference, with all kinds of work, varies according to the different muscles affected, but the patient almost always complains of more or less considerable disturbance of his vision. When the paresis has reached a certain height, or has even increased to complete paralysis, it may remain stationary for a variable period; sooner or later, however, secondary contractures occur, which render the symptoms more marked, and may considerably retard recovery. Great variations are often observed in the amount of the disturbance, especially in those cases in which paresis is caused by central disease, as in tabes. The double vision in such cases frequently disappears for weeks, and then recurs. If improvement takes place, the absolute mobility of the eye usually first begins to be restored, the diplopia remaining for some time longer; the double images then gradually approximate, or only become visible at the limits of the field of vision, and ultimately entirely vanish; and with their disappearance the disturbances of vision, the feeling of giddiness, and the abnormal position of the head, pass away. The secondary contractures remain the longest. In incurable cases the deviation of the eye is persistent, since it is caused by the contraction of the antagonistic muscles, and permanent strabismus results.

The *duration* of paralysis of the ocular muscles is also very variable, depending greatly on the causes. Rheumatic paralyses are usually of short duration, lasting only for a few weeks, rarely longer. I have several times observed rheumatic paralysis of the sixth nerve recover under galvanic treatment in less than three weeks. Syphilitic paralysis may last for months or years, and yet at length recover. In many central paralyses, which also exhibit great fluctuations, the same thing occurs. Incurable cases are of course of unlimited duration.

Diagnosis.—In forming the diagnosis the particular *muscles and nerves that are attacked* must first be ascertained, and the principal points on which reliance can be placed for this purpose are contained in the above-given account of the symptoms. Complicated cases, however, sometimes occur, in which several muscles or both eyes are affected, and which are therefore extremely difficult to diagnosticate. On these points we must refer to the elaborate work of A. von Graefe, and other special treatises.

Secondly, it is important to discover *the nature of the primary lesion*, though this is not always an easy task; the determination must be founded on general pathological principles, regard being paid to the causes, the course of preceding diseases, the accompanying symptoms, and other points.

The diagnosis of the *locality* of the disease is often much more difficult. It must first be determined whether the paralysis is of centric or of peripheral origin. This is only easily accomplished when the injurious influences are accurately known, or when other symptoms are present which facilitate the localization of the affection, such as cerebral symptoms, vertigo, sensory disturbances, hemiplegia, spinal symptoms, etc., or when there are symptoms of some disease in the orbit or at the base of the cranium, etc. Where such symptoms are absent, and particularly in isolated paralyses, we must be content with more or less probability. Unfortunately, electric treatment does not here possess any diagnostic value, because we at present possess no means of stimulating with certainty and without danger the several ocular muscles and their nerves. In this respect the following points may be attended to: When the seat of the disease is peripheral the paralysis is more often complete than when it is central; in peripheral paralyses the extent of fusion is generally greater than in central paralyses. The occurrence of galvanic hyperæsthesia of the portio mollis is in favor of a central origin.

When it is probable that the seat of the lesion is peripheral, it is further necessary to determine at what point in its course the nerve is affected, whether the lesion is at the base of the brain or in the orbit. The existence of symptoms resulting from the affection of other nerves either of the brain or of the orbit, and the etiology of the disease, often afford aid in the diagnosis. In paralysis of the third pair, the circumstance that particular branches are affected, the implication or otherwise of the pupil and of the power of accommodation, may all prove of importance in determining the exact seat of the disease.

But even where the seat of the lesion has been ascertained to be central, it may still have very various positions, which must be deduced from the general rules of diagnosis for cerebral diseases; on this point the occurrence of facial paralysis or hemiplegia on the same or on the opposite side, the existence of aphasia, and the evidence of bulbar paralysis or of some spinal affection, supply the necessary information. We can here only briefly refer to them.

The *prognosis* depends essentially upon the causes of the disease. It is almost invariably favorable in rheumatic cases, though even here recovery sometimes takes place with difficulty or not at all. Traumatic paralysis of the ocular muscles also usually pursues a favorable course. In paralysis from compression the prognosis must rest on the nature of the compressing cause. Only a doubtful prognosis can be given in syphilitic cases, since many of them do not yield to the appropriate antisyphilitic treatment. Paralyses, consecutive to diphtheria, almost always recover, whilst, on the contrary, those that are of centric origin are doubtful, and the prognosis must depend on the nature of the primary lesion. It is to be noted, however, that in incurable affections of the central organs, the prognosis of the accompanying paralysis of the ocular muscles may be relatively favorable, as occurs in tabes and in cerebral apoplexies.

Lastly, it may be observed that the prognosis of *complete* paralysis, which has lasted for some time, is much more unfavorable than that of incomplete paralysis of much longer duration. The prognosis is less favorable when in the course of treatment the absolute extent of excursion increases more quickly than the approximation of the double images; such cases heal slowly. It is favorable when a distinct and instantaneous improvement is effected by galvanic treatment.

Treatment.—It is unnecessary here to enter into any details in regard to the fulfilment of the causal indications, and it will be sufficient to remind the reader of the treatment that may be required for injuries, for rheumatic affections (such as warmth, diaphoresis, vesicants, iodide of potassium, etc)., for syphilis, for affections of the central nervous system, and especially for tabes and the like.

Amongst the direct remedies, the most important is *electricity*, which must be employed in accordance with the general principles that have already been given in detail. The mode of application must in many cases have reference not only to its direct anti-paralytic action, but to the fulfilment of the causal indications. Galvanic treatment is preferable to faradic, because, on well-known physical grounds, it is more certain that the necessary amount of the current reaches the deeper lying tissues.

Method of galvanization.—The stabile application of the current should be made in alternating directions, according to the position of the lesion—whether in the orbit, or in the middle or posterior fossa of the skull—either transversely through the temples or through the mastoid processes, by placing the electrodes in front of each ear, ultimately, also, longitudinally from the eye to the neck. The galvanization of the cervical sympathetic of the same side, appears, in some instances, to be serviceable (Benedict). In order to act directly on the paralyzed muscles, it is advisable to apply the anode to the neck, and to make the cathode glide over the closed eyelids, especially over those points which correspond to the paralyzed muscles. The currents used should not be stronger than may be sufficient to produce distinct contractions of the facial muscles when the face is stroked with the cathode. The application of the current should be of short duration, not exceeding two or three minutes, for example, and care should be taken to avoid too strong or sudden variations in the strength of the current (by interruptions or reversals). The results obtained by galvanic treatment are in many cases extremely favorable; generally a distinct and instantaneous improvement is exhibited, as shown by the increase of the absolute mobility of the eye and the approximation of the double images. The amount and duration of this improvement determines the quicker or slower rapidity with which recovery will take place. Much patience is often necessary in pursuing galvanic treatment, and many cases require to be galvanized for months before improvement occurs.

Methods of faradization.—This mode of applying electricity may be effected by means of direct faradic stimulation in the vicinity of the attachments of the affected muscles, either through the closed lids by means of a small sponge electrode, or through the conjunctiva by means of a fine brush acting as an electrode. The iris, when affected with mydriasis, has also been directly treated through the sclerotic by small electrodes; this, however, is a painful proceeding. The current must not be too strong, nor applied for too long a period. Good results are unquestionably obtained in many cases from faradization.

Little remains to be said in regard to the action of other remedies; it has been empirically discovered that iodide of potassium is of great utility in many cases of unknown origin. Subcutaneous injections of strychnia deserve mention; and the calabar bean may be of use in cases of mydriasis. All these remedies may be employed coincidently with electricity.

It is very advantageous, especially in the slight forms of paresis, and in the final stages of paralysis, to adopt an appropriate system of gymnastics for the ocular muscles, partly by means of prisms, which aid in effecting fusion of the double images, and partly by regular exercises with objects, which should be slowly moved from the region of single vision towards the side of the double images, the patient endeavoring to preserve single vision as long as possible. In cases of mydriasis, strong closure of the eyelids having been observed to be associated with contraction of thepupil, the so-called "compressing exercises" (Kneifübungen) have been recommended as advantageous. Very similar in their action are the accommodation exercises for short distances, in cases where there is strong convergence of the visual axis.

For the removal of the visual disturbances, caused by the double images, prismatic glasses can rarely be employed, since the distance of the double images from one another is variable; and the prisms requisite for compensation are, for the most part, incapable of being used as glasses. Such prisms can only be employed in cases of slight paretic deviation, and are then a great help to the patient.

Operative procedures, such as setting back and bringing forward the muscles, can only be adopted when all possibility of recovery, or even improvement, is excluded; the operations above named must then be conducted according to the indications afforded by ophthalmology. As a palliative remedy for the discomfort arising from double images, a pair of spectacles, with one dull glass, may be worn before the eye.

b. *Paralysis in the Region of Distribution of the Motor Portion of the Trigeminus.—Paralysis of the Muscles of Mastication.—Masticatory Paralysis of the Face.*

C. Bell, l. c. p. 217. — *Romberg*, l. c. p. 59. 1851. — *A. Eulenburg*, l. c. p. 492. — *Benedict*, Electrother. 1868, p. 307. — *Bärwinkel*, z. Pathol. d. Trigeminus. II. Diplegia trigem. motoria. Arch. f. klin. Med. XII. p. 608. 1874.

The motor fibres of the fifth nerve are essentially distributed to the muscles of mastication (the masseter, temporal, external and internal pterygoids), but they supply, also, one muscle of the soft palate (the spheno-staphylinus), the tensor tympani (the branch to this muscle proceeding from the otic ganglion), and, lastly, the mylo-hyoid and digastric through the nervus mylo-hyoideus. The only paralyses that are important in practice are those of the muscles of mastication.

Paralysis of the muscles of mastication is, upon the whole, of rare occurrence, as the nerves, on account of their deep position, are protected from external injury and cold. Accordingly the lesions which affect the motor portion of the fifth nerve are generally intra-cranial and situated at the base of the skull, such as periostitis, exostoses, caries of the bones, extravasations, aneurisms, and tumors of all kinds implicating the third division of the fifth, either inside or outside the skull ; lastly, cerebral affections, and, in particular, affections limited to the pons and the medulla oblongata, are those which lead to paralysis of the muscles of mastication on one or both sides. Thus double paralysis of these muscles has been repeatedly observed as a symptom of certain forms of so-called bulbar paralysis. Bärwinkel has recently described a case of this kind, and I have myself seen several.

The *symptoms* of this form of paralysis are very simple. If it be unilateral, there is difficulty or impossibility of performing the acts of mastication on that side; the patient chews exclusively on the healthy side. No contraction of the masseter or temporal muscles is perceptible, either to the sight or touch, though this is very distinctly marked on the healthy side by a periodically recurring hardness and swelling. Lateral movements of the jaw towards the sound side are rendered impossible by the paralysis of the pterygoids. In bilateral paralysis, so long as paresis only is present, fatigue is quickly experienced on mastication ; the patient gradually becomes incapable of finely breaking up meat, or dry hard bread ; he has to rest frequently while chewing, and at length, when the muscles refuse to perform their function, the lower jaw falls relaxed and powerless. In some instances a high degree of atrophy of the muscles occurs, which is easily recognizable to the touch by the depressions in the region of the muscles of mastication. Secondary contractures may also make their appearance in the paralyzed muscles, and thus cause the lower jaw to be permanently elevated.

The electric relations of the paralyzed muscles (which can only be made to contract with difficulty and by direct stimulation) have not been sufficiently investigated. In a case evidently of peripheral paralysis, I found both the faradic and galvanic excitability abolished. In bulbar paralysis there is a moderate decrease of excitability. Bärwinkel found in his case that three months after the commencement of the disease the excitability for faradic currents was lost, whilst it was preserved, though diminished, for galvanic currents.

Paralysis of neighboring cerebral nerves, as of those supplying the muscles of the eye, the facial nerve, etc., not unfrequently accompanies paralysis of the muscles of mastication. Very commonly, also, there is anæsthesia of the fifth pair, frequently limited to the third division. More rarely there are disturbances of taste, affecting the anterior half of the tongue. In addition, in many cases, there is a great variety of symptoms proceeding from the primary disease, amongst which the masticatory paralysis occupies a subordinate position. Nothing is at present known in regard to the effects of the paralysis of the spheno-staphylinus, and too little attention has been paid, as it appears, to the tensor tympani, since the lately published results of Lucae's investigations[1] indicate the possibility of diagnosticating paralysis of this muscle by the existence of abnormal acuteness of hearing for high sounds and subjective deep roaring sounds.

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[1] Berl. klin. Woch. 1874, Nos. 14, 16, 17.

The *diagnosis of paralysis of the muscles of mastication* presents no difficulty, if moderate attention be paid to the symptoms; the passive mobility with active immobility of the lower jaw, the absence of the characteristic swelling of the masticatory muscles when in action, and the mode in which the act of mastication is disturbed, scarcely admit of an erroneous interpretation. The prognosis rests entirely on the nature of the primary cause.

This must also be carefully considered in determining the treatment; but, in addition to the causal treatment, local application of electrical currents—intra-muscular faradisation or galvanization—may be recommended.

c. *Paralysis in the Region of Distribution of the Facial Nerve.—Mimetic Facial Paralysis.—Hemiplegia and Diplegia Facialis.—Prosopoplegia.*

N. Friedreich, de paralysi musculor. facici rheumat.—Progr. 1797. (Germ. trans. in "N. Friedreich's verm. med. Abhandl." Würzb. 1824.)—*Ch. Bell*, phys. u. path. Untersuch. 1832.—*Gaedechens*, Nervi facial. physiol. et pathologia Diss. Heidelb. 1832.—*Landouzy*, l'hémiplégie faciale chez les nouveau-nés. Paris, 1839.—*Bernard*, de l'alteration du gout dans la paral. du nerf facial, Arch. génér. de Méd. Dec. 1843.—*Marshall Hall*, Lancet, May, 1843.—*Landouzy*, de l'éxaltat. de l'ouie dans la paral. du n. facial. Gaz. méd. de Par. 1851, Nos. 6, 7.—*Ph. H. Wolff*, Oxyokoia durch Facialparalyse. Deutsch. Klin. 1851, No. 22.—*C. Davaine*, sur les paral. génér. ou partielles des deux nerfs fac. Gaz. méd. de Par. 1852, Nos. 46—50; 1853, Nos. 2, 3.—*B. Schulz*, Paralys. nervi fac. excentrica, etc. Wien. med. Woch. 1857, No. 44.—*Ziemssen*, Lähmung von Gehirnnerv. an d. Basis cerebr. Virch. Arch. XIII. 1858.—*Baierlacher*, Bayr. ärztl. Intell. 1859.—*Schulz*, differ. elec. Verhalten d. Musk. bei Faciallähm. Wien. Woch. 1860, No. 27.—*O. v. Grünewaldt*, über Faciall. Petersb. med. Ztschr. III. 1862.—*Wachsmuth*, progress. Bulbärparalyse u. Diplegia fac. Dorpat. 1864.—*W. Sanders*. Paralys. of the palate in fac. palsy. Edinb. med. Journ. 1865. Aug.—*Ziemssen*, Electr. in d. Medic. 3. Aufl. 1866.—*Bazire*, Case of facial paralysis, etc. Brit. med. Journ. 1867. Sept. 21.—*Trousseau*, med. Klin. des Hotel Dieu. Deutsch v. Culmann. II. Band. 1868.—*M. Rosenthal*, Charakteristik der verschiedenen Arten von Gesichtslähm. Wien. m. Presse, 1868.—*Pierreson*, de la diplégie faciale. Arch. génér. 1837.—*Bärwinkel*, Casuist. d. doppelseit, Faciallähmung. Arch. d. Heilk. 1867.—*Erb*, z. Pathol. periph.

Paralysen. Arch. f. klin. Med. IV. V. 1868.—Casuistik: zwei traumat. Facialisl. ibid. VII. 1870.—*Brenner*, Unters. u. Beob. z. Electroth. II. 1869.—*Lucae*, über Gehörstörung bei Facialisl. Verh. d. Berl. med. Ges. I. 1866.—*Hitzig*, z. peripheren Facialisl. Berl. klin. Woch. 1869, No. 2.—*Tillmanns*, Facialisl. bei Ohrkrankheiten. Diss. Halle, 1869.—*Hitzig*, Auffass. einiger Anomal. d. Muskelinnervation. Arch. f. Psych. u. Nerv. III. 1872.—*Huguenin*, über die cerebr. Lähm. des Facialis. Correspondenzbl. für die Schweiz. Aerzt. 1872, Nos. 7—9.—Compare, in addition, the treatises on diseases of the nerves by Romberg, Hasse, A. Eulenburg, M. Rosenthal, and others, and the various treatises on electrotherapeutics.

Facial paralysis is one of the commonest forms of paralysis. Scarcely any other nerve of the human body is so often independently attacked as the facial. This is partly attributable to its exposed position rendering it liable to various forms of injury, partly to its long course through a narrow osseous canal, and to its close proximity to organs very liable to disease, and partly to its long course in the central organs of the nervous system, so that the diseases of these parts are very frequently accompanied with that of the facial.

Facial paralysis is sometimes unilateral and sometimes, though rarely, bilateral; it may occur as a paresis, or more frequently as paralysis; it may be complete, and affect all the branches of the nerves, or it may attack particular branches alone. The remarkable mimetic disturbances and deformities, the impairment of various important and commonly employed movements accompanying paralysis of the facial, render this disease very tedious, and at the same time easily recognizable, both by its objective and subjective symptoms. If this be sufficient to account for the great interest which facial paralysis has always excited, that interest is still further augmented by the fact that facial paralysis is of the greatest importance in solving numerous neurological problems, since the facial nerve has been found to be a bundle of nerves of extremely different physiological functions. In consequence of the limited space at our disposal, the following very condensed statement is scarcely sufficient to afford an exhaustive picture of the numerous and important results which have been laid down in the very voluminous literature of facial paralysis, and which have been augmented and corroborated by our own personal experience.

Etiology.—There is no such thing as a decided predisposition to facial paralysis; at least the cause of many persons being frequently attacked by "rheumatic" facial paralysis admits of no clear explanation. We are only acquainted, therefore, with direct causes. Amongst these the most frequent and important is "catching cold." It has been established beyond a donbt that there is a close connection between the action of cold upon one side of the face, and the occurrence of facial paralysis, either immediately or after the lapse of only a few hours, or, more rarely, after several days. The histories of cases reported show a countless number of ways in which cold may produce these "rheumatic" facial paralyses—or, better, facial paralyses "from chill" (looking out of windows on a windy day, travelling in a railway carriage with an open window, sleeping near a damp cold wall, draught on one side of the face, etc.). Nothing certain, however, is known in regard to the real seat and kind of the anatomical changes leading to these paralyses; but it is now generally admitted that there is slight exudative inflammation and swelling of the neurilemma in the Fallopian canal; in slight cases the exudation is believed to be serous, in severe and protracted cases, plastic (Schultz, Bärwinkel, and others). In my opinion only one kind of anatomical change, probably slight inflammatory swelling of the nerve sheath, is present in all such rheumatic cases, the difference in locality causing the different degrees of severity of the attack. If the trunk of the nerve external to the Fallopian canal be affected, the motility alone is impaired, and compression is not sufficiently powerful to cause complete degeneration of the nerve. But if the nerve be attacked within the canalis Fallopiæ, it undergoes strong compression; degeneration is unavoidable, on account of the limited space, and a severe form of paralysis is produced. It is obvious that the different *intensity* and duration of the inflammatory or rheumatic process can modify this, which is only a general expression of the case. Furthermore, the extent of nerve affected within the Fallopian canal must produce very definite varieties in the general aspect of the disease, which can be made use of for the local diagnosis.

Facial paralysis is not unfrequently the result of *injury;*

thus it has been observed after a severe box on the ear, after a blow given by an ox, the horn of which penetrated the region of the ear (Bell), after various wounds, such as gun-shot injuries, fractures of the skull, fractures of the temporal bone, wounds due to missiles entering the internal ear, etc. Facial paralysis very frequently occurs after surgical operations about the face and region of the ear, especially after extirpation of the parotid gland; and, lastly, it is sometimes caused in new-born infants by the pressure of the forceps (Landouzy, Osiander).

Compression of the whole nerve, or of some of the branches, by affections of the parotid gland and adjoining parts may occasion facial paralysis; thus abscesses, inflammation, the formation of tumors in the parotid, swollen lymph glands behind the recesses of the jaw, deep-seated ulcerations and cicatrices—such, for example, as occur after scrofulous abscesses in the glands—have all been noted as causes.

Various *diseases of the ear* may also cause facial paralysis. Craig gives an example of this form of paralysis which was occasioned by the accumulation of hardened cerumen in the auditory meatus, the removal of which was followed by recovery. Suppurative otitis interna, followed by caries of the temporal bone, is, however, the most frequent cause of facial paralysis. This has been observed in innumerable cases, and may be explained partly by suppurative softening and destruction of the trunk of the facial nerve, and partly by secondary compressing neuritis, produced by the carious process in the Fallopian canal. The great extent throughout which the facial nerve lies in close proximity to the tympanic cavity, sufficiently explains the frequency of this connection. It can be easily understood, also, that bony tumors and neoplastic formations of all kinds, proceeding from the internal ear, occasionally compress and paralyze the facial nerve. It appears to be less certain that there is any connection between simple and suppurative catarrh of the middle ear and facial paralysis. Tillmann zealously espouses the opinion advanced by various other authors (Wilde, Deleau, Tröltsch, and others), that catarrhal diseases of the middle ear very frequently implicate the facial nerve, and thus occasion paralysis, and he believes that many cases which are usually described as rheumatic, result from a catarrh of the tympanic cavity; he has sought to prove the possibility of such intimate relations between the two, by citing a number of cases, and by referring to the close proximity of the nerve to the tympanic cavity, it being separated from the latter by only a very thin osseous lamella, or even only by fibrous tissue, and also to the fact that the nerve sends two branches into the tympanic cavity, and has the same nutrient artery. The course of these cases, the antecedent and concomitant disturbances of hearing, as well as the objective condition of the ear, sufficiently differentiate them from simple rheumatic cases.

A not unusual cause of facial paralysis is syphilis, the products of which, as gummata, periostitis, meningitis, exostoses, etc., at the base of the skull, in the temporal bone, or in the brain, very frequently affect the facial nerve. Syphilitic affections at the base of the brain in particular implicate the facial with other nerves.

Very various intra-cranial forms of disease may be accompanied by facial paralysis, and the following groups may be distinguished : in the first place, basal paralyses, originating from disease or compression of the nerves at the base of the skull (such as aneurisms, exudations, tumors of all kinds), and presenting the character of peripheral paralyses. Secondly, paralyses in the region of the facial nucleus, of the medulla and of the pons, which are most frequently caused by chronic inflammatory processes (progressive bulbar paralysis, multiple sclerosis), or by tumors and small apoplexies, or centres of softening. Thirdly, paralyses of the facial fibres in their course through the crura cerebri, along the central ganglia and in the corona radiata ; to these belong the very common apoplectic and ischæmic paralyses, which are usually connected with more or less hemiplegia, and are rarely quite independent and limited to the facial (Duplay). Lastly, paralyses from disease of the motor centres in the gray substance of the hemisphere, in very diverse affections of the cortical substance, such as abscesses, tumors, diffuse inflammation, etc. To this class belongs Hitzig's case, which has been so frequently quoted,[1] and probably also the case recorded by Leared;[2] to this group belong also those pareses in the muscles supplied by the facial nerve, which are not unfrequently to be observed in diffuse affections of the cortex, in dementia paralytica, etc., and betray themselves especially when an attempt is made to draw back the lips, and in similar movements.

Facial paralyses are rare in affections of the spinal cord, but they nevertheless occur when the disease extends as high as the calamus scriptorius, to the nucleus of the facial nerve, which is often the case in paralysis ascendens acuta. In tabes dorsalis, facial paralysis frequently occurs with partial paralysis of the oculo-motorius, atrophy of the optic nerves, etc. Facial paralysis has been occasionally observed after acute diseases, such as typhoid fever, scarlet fever, measles, and diphtheria; it has been observed also after some violent mental emotion, as intense fright.

Symptoms.—It often happens that the symptoms of facial paralysis appear quite suddenly, the patient awakening with it in the morning, or accidentally discovering it in the looking-glass, or being told of it first by a friend. More rarely it develops very gradually, spreading from branch to branch, and progressing from the slightest paresis to complete paralysis. Premonitory symptoms are often observed for several hours or for days, so that after pains in the vicinity of the ear and in the affected half of the face, or in the ear itself, subjective noises in the ear, deafness, and, in many cases, abnormal gustatory sensations (metallic, acid, cool taste) in the affected half of the tongue, have been experienced for some time, the symptoms of facial paralysis supervene more or less suddenly.

The symptoms are in the highest degree remarkable, and, when the paralysis is unilateral and complete, immediately strike the eye. The paralyzed side of the face appears smooth, flaccid, expressionless, without lines, wrinkles, or furrows, "as though ironed out;" the eye is widely opened and waters; the angle of the mouth is depressed on one side, is slightly open, and allows the saliva to escape; the whole mouth is drawn obliquely towards the healthy side, and the tip of the nose is turned to the same side; every stimulus to mimetic movements makes the aspect more striking; the sound side is alone brought into action; the patient laughs, weeps, speaks, and is angry only with the healthy side, the other remaining always immovable; the face is distorted in the most remarkable manner, because the paralyzed muscles yield to the traction of the healthy ones, and the more the patient strives to avoid grimaces the more marked they become. No voluntary movements can be performed by the muscles innervated by the facial nerve; no frontal furrows appear nor any corrugation of the eyebrows; the eyelids cannot be voluntarily closed; the palpebral fissure remains wide open (lagophthalmus), the lower eyelid not unfrequently separating from the eye and ultimately becoming ectropic. On attempting to close the lid, the upper eyelid falls, to some extent, partly in consequence of its weight and partly because the levator is relaxed; the globe is rotated inwards and upwards or outwards and upwards, and the pupil is thus brought beneath the upper eyelid; the fissure of the lids, however, remains wide open (in peripheral paralyses even in sleep, which is an important symptom, distinguishing them from facial paralyses of cerebral origin, in which the branches distributed to the orbicularis palpebrarum are not coincidently paralyzed). This inability to close the lid, and especially the coincident paralysis of Horner's muscle, interferes with the entrance of the tears into the lachrymal canals, and they continually overflow upon the cheeks (epiphora). The imperfect closure of the lids renders the removal of particles of dust from the eye impossible; and these particles, together with the stimulus of the air on the persistently open eye, cause hyperæmia of the conjunctiva, which may increase to catarrhal inflammation, and is not infrequently accompanied by inflammation and ulceration of the cornea and impairment of vision.

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[1] Facial paresis with abscess in the Sylvian fissure (?) (Klappdeckel) of the opposite side. Archiv für Psychiatrie, Band III., p. 231.

[2] A case of Paralysis of the right facial and abducens, in consequence of a cerebral abscess occurring several weeks after a blow on the left parietal bone. *Lancet*, 1869, March 6th.

The patient loses the power of wrinkling up the nose on the affected side, of raising the upper lip, of laughing, and of drawing the angle of the mouth up on that side. The lips cannot be pursed up, because the paralyzed side drags behind; whistling becomes impossible, and when any attempt is made to blow out the cheeks the air escapes on the paralyzed side, as does also the water when the patient tries to drink. Speech is rendered indistinct, because the labial sounds are imperfectly formed, and the necessary movements of the cheeks cannot be made. The movements of the cheek accessory to mastication also fail, and mastication is rendered difficult because the food lodges between the teeth and the cheek, and has to be brought back again by the finger. The external muscles of the ear are also immovable, though it is only in occasional instances that the failure of their function can be ascertained with certainty and facility; now and then the coincident paralysis of the platysma, of the posterior belly of the digastric and that of the stylo-hyoid can also be demonstrated.

Special attention is due to various other symptoms, which depend partly on the affection of branches of the facial proceeding from a higher level, partly on the implication in the disease of various tissues near the nerve,—symptoms that are only present in certain cases, but which have important bearings on the diagnosis and prognosis of these cases. We must describe these in some detail.

Behavior of the velum palati and uvula.—The velum palati has been observed to be affected with paralysis in a number of cases. When affected it hangs loosely downwards on the paralyzed side, occupying a lower position than on the sound side, and exhibiting decidedly diminished action in phonation and on reflex irritation, so that it is retracted towards the sound side. Difficulty of swallowing is often present. The speech acquires a nasal twang, and, on drinking, fluid escapes through the nostril of the paralyzed side. The uvula is not unfrequently oblique, the point being drawn sometimes to the healthy, sometimes to the paralyzed side, the cause of the difference, notwithstanding all the researches that have been made and the discussions that have been held in regard to it, not being yet perfectly understood. It is well known that motor fibres pass from the intumescentia gangliformis of the facial through the nervus petrosus superficialis major to the spheno-palatine ganglion, and from this to the velum palati; these serve for the innervation of the several muscles of the velum palati (chiefly the levator palati, but probably others also). If the cause of paralysis be situated above the intumescentia gangliformis, the fibres distributed to the velum palati are implicated, and consequently paralyzed. The vertical shifting of the position of the velum palati, particularly insisted on by Sanders, is easily explicable as a result of the implication of the levator palati. The abnormal position of the uvula is more difficult to explain, because this deviates sometimes to the paralyzed and sometimes to the healthy side. The deviation to the healthy side is easily explained by the preponderating influence of the healthy muscles, and Sanders has endeavored to explain the deviation to the paralyzed side by the unopposed action of the healthy pharyngo-palatine muscle. Moreover, it might be expected, from the manifold interweaving of the muscular fibres in the velum palati, and their diverse nervous supply, that great individual differences would occur, finding their expression in different positions of the uvula. But since the uvula diverges, to one or the other side, very commonly in healthy persons, little stress can be laid upon this symptom, and we must regard the paresis of the velum palati and its deviation in phonation only as evidence of the affection of the nervus pretrosus superficialis major.

Behavior of the sense of taste and the salivary secretion.—These, not unfrequently, present interesting and diagnostically important anomalies. It is well known that the chorda tympani, which proceeds from the facial nerve in the lower part of the Fallopian canal, is the chief agent in the sense of taste for the anterior half of the tongue, and the principal secretory nerve for several salivary glands. We have already, in speaking of the gustatory neuroses (p. 239, et seq.), sought to establish that the gustatory fibres of the chorda tympani accompany the facial for only a part of its course, probably leaving it by the nervus petrosus superficialis major, at the level of the ganglion geniculi. Hence, if the cause of the paralysis affect the facial nerve at any point between the ganglion geniculi and the giving off of the chorda tympani, disturbances of the sense of taste will occur, whilst such disturbances are absent in lesions affecting the nerve at the basis cranii, or below the giving off of the chorda. This conclusion is sufficiently supported by experience. In the first place, in many instances (Roux, Bazire, and others), a subjective metallic or sourish taste is experienced on the anterior half of the tongue of the same side, which frequently precedes the occurrence of the paralysis; careful testing then usually shows that the sense of taste is completely abolished, or materially retarded (especially for acid, sweet, or saline flavors) in the anterior two-thirds of the half of the tongue corresponding to the paralyzed side. Since the other half of the apex of the tongue, and both sides of the base of the tongue preserve their gustatory sensibility unimpaired, the disturbance is frequently unnoticed by the patient, and is only discovered by careful investigation. Whether the chorda also supplies sensory fibres to the tongue, the paralysis of which diminishes the sensibility of its apex, is at least doubtful. I have, hitherto, in uncomplicated cases, always observed that the sensibility of the tongue was unimpaired, though there was paralysis of the gustatory sense.

A diminution of the salivary secretion on the paralyzed side is less certainly and easily ascertained, yet it has been repeatedly demonstrated, first, by Fr. Arnold (1838), subsequently by Romberg and others, and the patients not unfrequently complain of an unusual dryness of this side of the mouth, "that the water does not flow on that side." The secretory fibres of the salivary glands leave the facial, partly with the chorda, partly by the petrosus superficialis minor; they appear to leave the brain in the trunk of the facial—at least Wachsmuth considers the dryness of the oral cavity as a constant symptom in paralysis having its seat above the ganglion geniculi.

Disturbances of the hearing are uncommonly frequent accompaniments of facial paralysis, but have very different relations to the paralysis, in different instances. From the purely accidental coincidence of facial paralysis with the common lesions of the ear, a causal connection between facial paralysis and auditory disturbances may occur in the following ways: 1. There are certain diseases of the middle ear, and of the adjoining parts of the temporal bone, which either depend upon the same lesion as the facial paralysis, and are co-effects of the same cause (wounds, "catching cold," etc.), or from which the morbid process has extended to the nerve. In these cases, in addition to the difficulty of hearing and subjective noises, objective indications of the diseases in question, such as the presence of exudation in the tympanic cavity, perforation of the tympanic membrane, suppurative aural catarrh, and frequently galvanic hyperæsthesia of the auditory nerve, may also be demonstrated. 2. In addition to the paralysis of the facial nerve, there is a similar affection of the auditory nerve, which indeed cannot easily entirely escape being affected by diseases which affect the facial at the base of the brain and in the meatus auditorius internus. In these cases, whilst investigation shows that the middle ear is intact, there is difficulty of hearing, or complete deafness, with subjective noises and occasionally abnormal conditions of galvanic excitability of the auditory nerve. 3. Lastly, paralysis of the facial nerve, with complete absence of the above-named changes, may occasion disturbances of audition, which have recently been satisfactorily explained. Roux first observed in his own case a disagreeable sensation in his ear with loud noises. Wolff then described and sought to explain "oxyokoia" occurring in cases of facial paralysis; this symptom has become well known through Landouzy's treatise, and has since been established by various authors, whilst it has lately been made the subject of special communications by Lucae and Hitzig. According to the researches of Lucae [1] there can be no longer any question that the hyperacusis (or abnormal acuteness of hearing of all musical tones, which may also express itself as an abnormal power of perceiving deep notes, which is often associated with a subjective sound of high pitch) present in facial paralysis is referable to paralysis of the stapedius muscle and the resulting overaction of the tensor tympani. If this symptom of abnormal acuteness of hearing be present, we are therefore justified in believing that the lesion is situated in the facial nerve above the point where the little branch for the stapedius muscle is given off.

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[1] Berl. klin. Woch. 1874, Nos. 14, 16, 17.

Disturbances of the sense of smell have not been very unfrequently observed upon the paralyzed side, though they stand in a quite indirect relation and indeed two-fold connection with the facial paralysis; on the one hand, on account of the insufficient discharge of tears, there is an abnormal dryness of this side of the nose which impairs the sense of smell, and on the other, the paralysis of the muscles moving the alæ nasi (levator alæ nasi and compressor narium) seriously interferes with the easy access of the air to the olfactory canal of the nose in the mode that has been already described in the account given of anosmia. (See above, page 258, et seq.)

The behavior of the tongue appears to be completely normal, even when most carefully tested in all uncomplicated cases. When protruded, it is straight, and it lies straight in the floor of the oral cavity. The abolition of the motility of the digastric and the stylo-hyoid muscles appears to exercise no noteworthy influence on the position and movement of the tongue. The statements in regard to the obliquity of the tongue in isolated paralysis of the facial appear consequently to depend in great part upon an easily explicable error of observation; for, on opening the mouth, its paralyzed angle is drawn towards the healthy side, and is consequently considerably nearer to the border of the tongue than the healthy angle is, and thus the tongue may present the appearance of being in an oblique position.

The *sensibility* of the paralyzed half of the face as a rule remains normal, providing the trigeminus is unaffected. Considering the intimate association that exists between the fibres of the fifth pair and those of the plexus anserinus, this is very noticeable and indicates that the seat of the paralysis is usually in the trunk of the facial. If the peripheral branches be affected, corresponding disturbances of the sensibility may usually be observed. In regard to the relations of the *muscular sensibility* (electro-muscular sensibility), I have not been able to find any satisfactory statements, though it is unquestionably deserving of being carefully examined and tested.

The *effect upon the reflex movements* varies considerably in different instances; they are completely abolished in the paralyzed muscles in all cases of peripheral origin. The few faint attempts that have been made to speak of some reflex actions being present in peripheral paralysis must remain unheeded, so long as no irrefragable proofs are advanced of their existence. In these cases no reflex actions can be obtained by stimulation of the conjunctiva or visual apparatus or of the skin of the face. The contrary obtains in cases of paralysis proceeding from the encephalon and especially from the hemispheres. The reflex actions are here completely preserved, and the eyelids, which the patient is entirely unable to close by a voluntary effort, readily blink and close on irritation of the conjunctiva and during sleep. Benedict has laid great stress upon the occurrence of unusual and especially upon decussated reflex actions as a means of diagnosticating the seat of the disease in the neighborhood of the facial nucleus ; but reliance is only to be placed upon them when all precautions have been taken especially to avoid diffusion of the current when electrical irritation is employed. If this be done, we are justified in admitting the presence of a disease of the gray substance in the neighborhood of the facial nucleus.

What has been said in regard to the reflex movements, may be repeated for the *associated movements :* in peripheral paralyses they completely fail ; in central paralyses they may be present, and it will depend upon the seat of the morbid process in the brain whether the muscles withdrawn from the influence of the will contract in the ordinary manner in the movements of respiration, in yawning, and in laughing. Important points in regard to diagnosis may be determined thereby.

As a rule, *no vaso-motor disturbances* are observed in facial paralysis—at least, nothing of the kind is to be perceived in the skin ; it is even frequently mentioned in the reports of cases of facial paralysis, that blushing occurred in the same manner upon the affected as upon the healthy side. The hyperæmia of the conjunctiva is only a consequence of stimuli acting constantly on the unprotected globe. This, however, does not exclude the possibility that considerable vaso-motor disturbances may occur in the muscles supplied by the facial. Such changes are, indeed, highly probable, since in many cases, especially in the so-called severe rheumatic and traumatic paralyses of the facial, as well as in paralysis of this nerve from compression, well-marked *trophic* *disturbances* are frequently observed. There is, for example, a high degree of atrophy of the muscles, which in protracted and incurable cases may lead to their complete disappearance. The paralyzed half of the face then appears greatly sunken; the skin, which is as thin as paper, is drawn tightly over the bones—the contours of which are brought strongly into relief; and the lips become thin and flabby. On the other hand, no change is observed in the external skin, the hairs, or epidermis.

Lastly, the *electrical behavior of the paralyzed nerves and muscles* is of the greatest importance from various points of view. It has been most profoundly studied, and its importance most accurately estimated, in the so-called *rheumatic facial paralysis*, and is deserving here of special attention. Careful electrical examination of a great number of cases shows that they may, according to their reaction with electricity, be divided into several groups, which differ from one another considerably in their course. In the first place, there is a group which *presents no change* in the faradic or galvanic excitability, either in the nerves or in the muscles. Throughout the whole duration of the disease, the paralyzed nerves and muscles react quantitatively and qualitatively exactly in the same manner to both kinds of currents as do the non-paralyzed. *The prognosis in all such cases is extremely favorable.* They recover in the course of two or three weeks, either spontaneously or under any kind of treatment. This is the *mild form* of rheumatic facial paralysis. (Brenner only observed in one case, that recovered in a few days, a slight diminution of faradic and galvanic excitability after the second day, which, however, soon again disappeared.)

Those facial paralyses of rheumatic origin, which exhibit in the most typical form all those quantitative and qualitative changes of excitability which we have termed the reaction of degeneration, may be united in a second group. It was in facial paralysis that this anomalous electrical reaction was first observed (Baierlacher), and it has been most frequently and carefully investigated in it; numerous observations have now sufficiently shown that the course of these changes of excitability is exactly the same in facial paralysis as in traumatic paralysis,

—in fact they present the typical character of the reaction of degeneration. We may therefore refer to the above-given detailed account (page 426) of this form of reaction, since what is there stated is applicable also to facial paralysis. There is diminution and loss of faradic and galvanic excitability of the nerves, loss of the faradic excitability of the muscles, quantitative increase and qualitative alteration of the galvanic excitability of the muscles, increase of their mechanical excitability—such are the principal characters of the condition which we need not here stop to describe in detail. It may, however, here be once more mentioned, that according to the more or less advanced recovery of the paralysis, the characters presented on electrical exploration may vary; but with the above-given statements before us, the stage of the disease will be easily discoverable. No doubt can be entertained that the above described (p. 412) histological changes in the nerve and muscles here also form the basis of the reaction of degeneration; at the same time, special investigations on this point are needed. The prognosis of this second group is essentially unfavorable. In almost all cases the duration of the affection must be expected to be considerable, lasting for two, four, six months, or even more, and such a case as that reported by Brenner, in which, the reaction of degeneration being fully developed, recovery was almost complete in the course of six weeks, must be regarded as exceptional. Traces of the paralysis are often observable after years, in a certain stiffness of the movements, slight contractures, and muscular twitchings. This is the severe form of rheumatic facial paralysis.

My own observations teach me that no complete and sharp distinction exists between these two groups in nature, but that there are transitional states, which I shall term intermediate forms. In these the reaction of degeneration is not only completely developed, but it presents the characters above described (p 437). In accordance with this, the prognosis of these intermediate forms, of which I have observed about six in a total of thirty-six cases, is relatively favorable; they recover in from four to six weeks, and the motility is often completely re-established at a time when the alterations of excitability are still strongly marked. The characteristic feature of this form is, that the faradic and galvanic excitability of the nerve is not completely lost, but only somewhat depressed, whilst the muscles—which always remain excitable through the nerve in the ordinary way—exhibit in the most exquisite form the increase and qualitative alteration of the galvanic excitability in addition to the increase of the mechanical excitability. Even at the close of the first week this simple depression of the excitability of the nerve may be observed, partly owing to the circumstance that, to produce the minimum contractions on the paralyzed side, somewhat greater strength of current is required than upon the sound side, and partly because with equal strength of current, both faradic and galvanic, the contractions are much less free on the paralyzed side. No further diminution, however, occurs, but with the re-establishment of motility, which usually occurs at an early period, the electrical excitability of the nerves gradually returns to its normal standard. In the meanwhile, the already mentioned alteration in the muscles becomes developed in the most distinct manner, in the course of the second, and, more rarely, of the third week, and exhibits, both on direct and on indirect excitation, a very different behavior. In direct iritation their contraction is slow and protracted ; the anodal closing contraction is stronger than the cathodal closing contraction, whilst on indirect excitation the contraction is short and quick and the cathodal closing contraction stronger than the anodal closing contraction. If, in addition, early restoration of motility occur whilst the above-mentioned partial reaction of degeneration persists for weeks, the symptoms are rendered still more interesting. For the practitioner, however, the prognostic significance of these symptoms is of chief importance. In a scientific point of view, it is interesting to observe that a characteristic alteration in the muscles may occur even when the nerves have not undergone complete degeneration ; but we do not yet possess any explanation of this interesting fact.

In *traumatic paralyses of the facial nerve*, such as may be occasioned by surgical operations, gunshot wounds, fractures of the temporal bone, etc., the reaction of degeneration exhibits itself in a typical form. This is the case also in all *paralyses from sufficiently severe compression*, such as may be caused by tumors of the basis cranii, caries of the temporal bone, neuritis, etc. If the compression of the nerve be only moderate, their electric excitability may remain completely normal. Brenner found in one case of paralysis, consequent on suppuration in the tympanic cavity, and in another case, following a suppurating tumor of the parotid gland, a considerable and uniform increase of both the faradic and the galvanic excitability of the nerves and muscles.

In paralyses resulting from diseases in the *vicinity of the facial nucleus*, as, for example, in progressive bulbar paralysis, a simple and moderate diminution of the electric excitability occurs in the nerve and muscles when the paralysis has lasted for some time.

When it is maintained, as it has been by M. Rosenthal, that in *diseases of the pons* the reaction of degeneration occurs in the region of distribution of the paralyzed facial nerve, the observation only holds good for those cases in which tumors in this region compress the nerve at the base of the skull, and thus really occasion a peripheral paralysis of the nerve.

In paralyses above the pons, that is to say, in purely cerebral and for the most part apoplectic paralyses, there is usually no change of the electric excitability. In some instances, however, a slight increase may be demonstrated to be present at a certain stage of the disease.

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A few words may here be added in regard to *bilateral facial paralysis*, the so-called diplegia facialis, which many authors have endeavored to distinguish as a special form of disease. But inasmuch as the two facial nerves, so far as our present knowledge extends, have no common point except the point of decussation, which is situated in the pons, the so-called diplegia facialis is as a rule only a more or less accidental coincident affection of the two nerves at some point of their course. It is obvious that this accident is the more likely to occur the nearer the two nerves are to each other, as at the basis cranii, in the medulla oblongata, and in the pons ; on the other hand, it occurs more rarely the further apart they run from one another, either in the direction of the periphery or towards the cortex of the brain. The observations that have been made upon the etiology of the affection are in accordance with this. (See Wachsmuth.) Diplegia Facialis is of most common occurrence in chronic affections of the medulla oblongata, and, as is well known, the bilateral or diffuse facial paralysis constitutes a characteristic feature in the symptomatology of progressive bulbar paralysis.

In the next place, affections of the pons (which are, however, upon the whole, rare) easily lead to diplegia of the face, partly owing to the implication of the fibres of both facial nerves in the pons itself, and partly from one facial nerve being paralyzed in the pons, and the other at the basis cranii (hence peripherally) by compression. Morbid processes at the base of the brain, especially the syphilitic ones, very commonly lead to diplegia of the face. On the other hand, it is rarely produced by true cerebral diseases (though a case has been recorded by Romberg and Magnus), and it may result from very diverse affections occurring at long intervals. Both facial nerves may be coincidently paralyzed peripherally, though in many instances the periods at which they are affected may be widely separated. Bilateral paralysis has been observed to arise from rheumatic causes, from wounds affecting both temporal bones, from bilateral otitis interna, and from caries of the temporal bones. Lastly, it is clear that extremely various complications of other kinds may occur, so that upon the one side apoplectic and upon the other rheumatic prosopoplegia may be associated; that upon the one side otitis interna, and upon the other a basal tumor may occur as causes of the paralysis. In view of these facts, it can never be considered sufficient to simply diagnosticate the existence of diplegia facialis, but in every instance the pathogenesis of the facial paralysis of both sides must be ascertained.

The phenomena of diplegia facialis are, *per se*, sufficiently clear, and do not easily escape attentive observation. The immobility which, in hemiplegia facialis, is present upon one side, here affects both sides to a greater or less degree, according to the nature of the particular case. The oblique position of the chin, mouth, and nose, the grimaces, are of course absent, but the face, especially in complete paralysis, at once attracts attention by the peculiar stiffness and fixedness which it preserves under all circumstances, even when the emotions are most strongly stirred; the patient "laughs and weeps as though behind a mask." When the disease is strongly marked, the lagophthalmus and lachrymation affecting both eyes, the difficulty which is experienced in moving the lips in speaking and laughing, the impairment of the articulation, the imperfectly performed movements of mastication, the incapacity of blowing, the escape of saliva and fluids from the mouth, the nasal tone of voice, and the difficulty of swallowing, all render the diagnosis of the disease easy. The other symptoms, such as the disturbances of the gustatory and auditory senses, will not be overlooked in certain cases, and from the concomitant symptoms in adjoining nerves and organs, and from the results of electrical investigation, taken in connection with the anamnesis, not only the seat, but the nature of the disease affecting each of the two facial nerves may, in every case, be easily determined.

Course and terminations.—The course of facial paralyses varies extremely in accordance with the diversity of the causes by which they are produced.

In paralyses of *rheumatic* origin, the onset is usually sudden, and is seldom accompanied by premonitory symptoms, such as pains in the head and face, auditory disturbances, etc. In most instances all the external branches, and in some cases, a part of the internal branches are affected. The apparently unchangeable picture presented by paralyses of this kind, in which often only electrical investigation affords us the means of recognizing the interesting processes of disease occurring beneath the surface, lasts for a very variable period. In slight cases, traces of motility may occur on the eighth, tenth, or twelfth day, and the recovery may be so perfect in the course of two or three weeks, that no traces of the antecedent disease are perceptible. The same may be said of cases of intermediate intensity, except that the recurrence of the motility takes place at a somewhat later period, and that complete recovery occupies a somewhat longer time. In such cases a certain stiffness of the face may remain for some weeks.

But if, on electrical investigation, it appears that the *severe* form is present, the recurrence of motility must not be expected for two or three months. The first traces of its re-establishment are, as a rule, perceptible about this period, and it progresses slowly, extremely slowly indeed, so that often two or three months more must elapse before recovery can be regarded as tolerably complete. It is in these cases that a very characteristic series of symptoms is presented, which delay recovery, and for a long time, even for many years, impress on the face the traces of the antecedent affection. I refer to the *secondary contractures and spasms* of the muscles, which have recently been very minutely described by Hitzig, although they were well known to the earlier observers (Duchenne, Remak, M. Meyer, Benedict, Erb, and others). These usually appear in the course of the third or fourth month in the form of a slight tonic contraction in the paralyzed muscles, usually making its first appearance at the angle of the mouth; the naso-labial fold becomes again visible, and distinctly marked. The angle of the mouth on the paralyzed side is drawn somewhat outwards and upwards, and remains fixed permanently in this position; the cheeks become more firmly pressed against the teeth, and oppose more resistance to extension, become more rigid; at a subsequent period the fissure of the eyelids becomes somewhat narrower, owing to contracture of the orbicularis palpebrarum, and thus the affected side of the face acquires a very peculiar and characteristic aspect. We entertain no doubt that this muscular rigidity has its principal cause in the histological processes in the muscles, which are found in paralyses accompanied by the reaction of degeneration, and which have been already described in detail at page 417 et seq. This much is certain, that this is not a condition of "electrical muscle tetanus" induced by electric treatment (though Remak attributes it to faradic, Duchenne to galvanic treatment), for it occurs in cases in which no electric treatment has been employed.

With the restoration of motility, spontaneous movements begin to make their appearance in the previously paralyzed muscles, in the first instance, in the form of scarcely perceptible contractions at the angle of the mouth, or around the eyes, which gradually so increase in intensity and frequency that they may ultimately be mistaken for a moderately severe convulsive tic. These contractions are in part quite spontaneous, and occur in this or that part when the patient is at perfect rest, most frequently as a rapid twitching in the muscles inserted into the angle of the mouth; they must also in part be regarded as associated movements, which occur very characteristically, on each effort of innervation directed to the muscles supplied by the paralyzed nerves. When an attempt is made to close the eyes, the angle of the mouth on the same side is drawn outwards and upwards; on attempting to raise the eyebrows the zygomatici contract, and when an effort is made to draw the angle of the mouth to one side, the fissure of the lids contracts, etc. As a rule, however, associated movements of this kind do not occur on innervation of the masticatory or of the brachial muscles. Lastly, these contractions occur in a reflex manner, with varying facility in different cases; thus, through the fifth pair, as a result of touching, pricking, or faradisation of the skin, or touching the cilia, and through the optic nerve, as by making a rapid movement towards the eye. In rare cases they increase to very strongly marked tonic and clonic spasms, which may occasionally extend to the facial of the other side, to the trigeminus, and even to more remote nerve regions. Hitzig thinks these symptoms are referable to an abnormal excitability of the medulla oblongata, which becomes developed in a still unknown manner, in consequence of peripheral facial paralysis. However obscure the pathogenesis of these conditions may be, they are characteristic results of severe rheumatic paralyses of the facial, as well as of those paralyses of this nerve that proceed from wounds and pressure. They often persist for a very long period, having been known to last for from eight to thirteen years, though they may also completely disappear in the course of a short time.

Traumatic, like severe rheumatic paralyses of the facial nerve, have, as a rule, a very protracted course. In favorable cases recovery takes place in the course of from three to six months, but it is frequently very incomplete, and the paralysis commonly remains for life. Slight traumatic paralyses may, on the other hand, undergo rapid recovery, as in the case, for instance, of the paralyses that occur in new-born children from the pressure of the forceps, and which, as a rule, disappear in the course of a few weeks.

In *paralyses from pressure* and in other peripheral paralyses, such as those resulting from otitis interna, from neuritis and syphilis, the course varies with the severity of the nerve lesion and with the nature and curability of the cause. Where the compression of the nerve has not been very severe, and where no alterations in the electrical excitability are demonstrable, a more or less rapid recovery, varying of course with the condition of the primary lesion, may be anticipated. Where, on the other hand, the existence of the reaction of degeneration shows that degeneration has taken place in the nerves, a protracted course may be looked for, and many cases prove incurable.

In *paralyses occasioned by disease of the central organs*, the course of the disease varies considerably, and depends entirely upon the nature and development of the primary disease, which has sometimes appeared suddenly, sometimes gradually. In many cases the paralysis is partial, since it is common for the branches distributed to the orbicularis palpebrarum and to the frontalis to remain intact. Frequently rapid recovery is observed, as, for example, in slight apoplectic cases, or it remains stationary, as in cerebral softening from embolism, tumors, etc. ; or, finally, it may progress steadily to a fatal issue, as in bulbar paralysis and carcinoma of the skull. But few general remarks can be made on this point, and further details would lead us beyond the limits prescribed for this work.

Diagnosis.—The existence of facial paralysis can, in general, be easily ascertained, and any error can only result from extreme carelessness. The only cases which can present any difficulty are those of very slight partial paresis, such as occurs in cortical affections, and at the very commencement of bulbar paralysis; but even here very slight grades of the functional disturbance may be recognized by careful attention to the play of the patient's features, by testing the finer and more complicated movements, as, for example, showing the teeth, pronouncing difficult words rapidly, whistling an air, etc. Paralysis of the facial is not, however, always recognizable in the new-born child, since its expressionless face does not appear materially altered when at rest. But even here the distortion that occurs on crying, the difficulty experienced in sucking, and the lagophthalmus during sleep, render it possible for the diagnosis to be correctly made.

The diagnosis of the *immediate causes* and *exact seat* of the paralysis is more important and more difficult, and demands careful investigation, and, as far as possible, accurate estimation of all the circumstances.

In the first place, an attempt must be made to discover the true *seat of the lesion.* The first point is to determine whether the paralysis has a peripheral or central origin. The following circumstances are more or less in favor of its peripheral origin: the affection of all the external branches, lagophthalmus, even during sleep, the absence of all reflex movements, the presence of the reaction of degeneration, atrophy of the muscles, symptoms of disease of the organs adjoining the peripheral part of the facial nerve, as of the parotid gland, the internal ear, the temporal bone, and the nerves at the base of the brain; clear evidence of a peripheral cause, as of a wound; and, lastly, the absence of all cerebral symptoms and of the implication of other cerebral nerves. Of course the more complicated conditions, tumors at the base of the brain, for example, require particular attention. In favor of a central seat are the following points: partial paralysis (some of the upper branches, in particular, which are distributed to the face, remaining intact), the fact that the eye can be closed during sleep, as well as voluntarily, the preservation of reflex acts, retained or even exalted electric excitability, the presence of other cerebral symptoms, vertigo, sensory disturbances, hemiplegia, weakness of the tongue, disturbance of speech, and considerable difficulty of swallowing.

This general localization is, however, insufficient; with our present experience we are even in a position, from the symptoms, to establish subdivisions with some degree of certainty, both in the peripheral and in the central course of the facial. The points to be relied upon for this purpose are in part derived from the affection of the branches given off by the facial, as the posterior auricular, the chorda tympani, the stapedius, and the nervus petrosus superficialis major, and in part from the implication of various nerves running in close proximity with the facial in the central organs, from the position of the decussation, and from the characters of the reflex acts. These subdivisions, of course with the necessary reserve, we shall here briefly sketch.

1. If complete paralysis of all the facial branches, due to some rheumatic cause, be present, if all reflex acts be absent, if the electric excitability be normal, if there be no disturbances, as of the taste or hearing, or of the velum palati, the *trunk* of the *facial* is affected externally to the *canalis Fallopiæ* (the subsequent proof that the auricular muscles—supplied by the nervus auricularis posterior—are unaffected will still further support this determination of the seat of the disease in the slighter forms of rheumatic paralysis).

2. Paralysis of all the external branches, including the auricularis posterior, rheumatic causes, reaction of degeneration, absence of gustatory disturbance, *indicate paralysis of the facial trunk within the canalis Fallopiæ and below the giving off of the chorda tympani* (this constitutes the most frequent form of "severe" rheumatic paralysis).

3. If the same symptoms be present, but with disturbance of the sense of taste, we may conclude *that the cause of the paralysis affects the trunk of the nerve between the point where the chorda tympani is given off, and the ganglion geniculi.* The giving off of the nervus stapedius permits a still further subdivision to be made: if abnormal acuteness of hearing be present, the lesion is situated above the origin of this nerve, as, for example, in a case reported by Ba e ; but if the acuteness of hearing be not present, then below it.

4. Paralysis of all the external branches, reaction of degeneration, abnormal acuteness of hearing, distnrbance of the sense of taste, *with* paresis of the velum palati (abnormal dryness in

the mouth), indicate that the *lesion is in the vicinity of the ganglion geniculi*, because into this ganglion the gustatory fibres enter, and from it the fibres of the velum palati emerge.

5. Paralysis of all the external branches, paresis of the velum palati, and dryness of the mouth, *without* gustatory disturbance; reaction of degeneration, or simple diminution of the electrical excitability; dullness of hearing, roaring in the ears, abnormal galvanic reaction of the portio mollis; implication of other nerves at the base of the brain,—collectively indicate *paralysis of the facial nerve at the base of the skull.*

The intra-cranial relations are both more complex and more uncertain.

6. If there be complete paralysis of the facial branches, paresis of the velum palati, absence of gustatory disturbance, simple diminution of electrical excitability, and especially if unusual or crossed reflex actions be present, *lesion of the facial nucleus* may be diagnosticated. The fact of the affection being seated in the medulla oblongata is rendered more certain by the implication of those cerebral nerves that have their origin in this part—the hypoglossus, accessorius, vagus, trigeminus, abducens, etc.

7. Complete paralysis of the branches distributed to the face, paresis of the velum palati, preservation of the reflex actions, the absence of any gustatory or auditory disturbance, normal electrical excitability, paralysis of the opposite extremities (*paralysie alterne*, Gubler), render it certain that the *seat of the lesion is in the pons* (at the same time alternate hemiplegia is not present in all affections of the pons, but only in those which affect it below the decussation of the facial). Moreover, the symptoms are often rendered very complicated in consequence of diseases of the pons exerting a peripheral compression of the nerves at the base of the cranium.

8. Partial paralysis of the facial branches (the superior ones remaining free), paresis of the velum palati, preservation of the reflex activity and electrical excitability, paralysis of the extremities of the same side, indicate that *the seat of the lesion is above the pons and situated in the crura cerebri and cerebral hemispheres.* The occurrence of crossed paralysis of the third

nerve with facial paralysis permits us to locate the disease in the peduncle itself.

It need scarcely be observed that it is rare in practice to meet with pure examples of these different seats of disease, the symptoms of which, moreover, require corroboration and rectification by repeated observation. All who possess some knowledge of the pathology of the nerves know how very complex conditions may arise by extension of the morbid processes, by the existence of separate seats or centres of disease, and by the accidental proximity of disturbing causes, as well as by the occurrence of several distinct forms of disease, so that erroneous conclusions can only be avoided by the exercise of the greatest caution, and by a thorough knowledge of the subject. This must, however, always be kept in view in order to avoid the possibility of error in the local diagnosis of facial paralyses.

If a positive conclusion in regard to the seat of the lesion has been arrived at, the solution of the question as to the nature of the disease is a matter of less difficulty. This must be based on general pathological grounds; any etiological and anamnetic conditions, complications of the most various kinds, and the results of electrical investigation, afford, as a rule, the means of drawing a positive conclusion in regard to the disturbance affecting any part of the facial nerve, and it is only rarely that we are in a position to enrich the history of "essential" paralyses by a new case. In many instances, the evidence pointing to a definite seat allows a conclusion to be drawn in regard to the nature of the lesion, whilst, conversely and not uncommonly, when the cause of the disease is known, we may form some idea of its seat.

The *prognosis* of facial paralysis must always essentially depend on the nature of the primary nerve lesion, that is to say, upon the cause of the paralysis, and may in part be deduced from what has been already stated. The seat of the lesion is of little importance; whatever may be the part of the facial nerve affected, some cases are curable and some are incurable. The most unfavorable are those resulting from the pressure of incurable tumors, from caries of the temporal bone, from fractures of the skull, and from gunshot injuries, and those also which proceed from bulbar sclerosis, tumors of the brain, etc. Those arising from apoplexy, cerebral embolisms, etc., are much more favorable, and usually disappear in the course of a few weeks. The prognosis of syphilitic paralyses is doubtful ; they are certainly not always curable, even when energetic anti-syphilitic treatment is practised. The prognosis in paralyses resulting from parotitis, otitis interna, etc., depends on the curability of these affections. As a rule, perfect recovery takes place after simple section of the nerve or of its branches.

The *prognosis of rheumatic facial paralysis* is of great practical importance, and is in general favorable, most cases recovering ; though some do not, or only incompletely. Contractures of the muscles and stiffness of the paralyzed half of the face, sometimes with spasmodic movements, often remain for a long time. Electrical investigation permits a very precise forecast to be made, at a tolerably early period, in regard to the duration of the affection. If the electrical excitability be completely normal at the end of the first week, recovery takes place in two or three weeks ; if at this period both the faradic and galvanic excitability be distinctly but only slightly diminished, recovery may be expected to occur in the course of from four to six weeks ; if, at the close of the first week, the electrical excitability of the nerve be already considerably depressed or altogether lost, the patient may anticipate that the disease will last for several months, and may calculate upon the subsequent occurrence of contractures. The further alterations of excitability that take place in the two latter cases, in the course of the second week, will serve to strengthen the prognosis.

Young persons appear to recover more rapidly from "severe" rheumatic forms than old ones do ; but there are exceptions to this rule. The "intermediate forms" I have, however, only seen in young persons, or those of middle age. According to my experience, it is incorrect to say that recent rheumatic paralyses of the facial nerve have always a favorable prognosis, whilst chronic cases have always an unfavorable prognosis. The treatment has little influence upon it. Slight cases speedily recover, even without any treatment, and in more severe cases judicious treatment, even when commenced very early, does not arrest the progress of degeneration or materially shorten the duration of the disease. No doubt can, however, be entertained that the prognosis of chronic cases is, on the whole, less favorable, and this depends on the circumstance that the slighter cases never become chronic, but quickly recover, whilst severe cases always become chronic, even when they are taken in hand at an early period.

Treatment.—The causal indications must first be attended to. Thus in those paralyses that are of cerebral origin, the appropriate treatment—which need not here be minutely detailed—must be prescribed. In syphilitic paralysis, mercury or iodide of potassium should be prescribed; whilst in otitis interna, caries of the temporal bone, and in fractures of the skull, the corresponding otiatric and surgical treatment must be adopted. Rheumatic forms of facial paralysis are more frequently the subject of treatment than any other, and great divergence of opinion exists in regard to the method of attacking the disease. Upon the one hand, absolute reliance is placed upon a multitude of remedies; and on the other hand, profound scepticism is entertained in regard to all successes reported. The occurrence of slight and severe forms readily explains these differences of opinion. The slighter forms really require no treatment at all, as they recover in a very short space of time, or can at most be somewhat more quickly caused to disappear by electricity than if left entirely alone. But since it is impossible, in the very first days, to distinguish with certainty the slighter from the more severe forms, and since a causal treatment, instituted at as early a period as possible, is followed by the relatively best results, it should be commenced as soon as the case is seen. In cases that fall under observation somewhat later, after the seventh day, and can certainly be recognized as belonging to the milder form, the causal treatment may be omitted, and electricity alone be employed.

Proceeding on the supposition that in rheumatic paralysis there is a slight inflammation accompanied by exudation, a kind of neuritis of the facial nerve, we should adopt those local and general measures which are usually employed for the removal of rheumatic inflammation; the patient should be directed to take one or more vapor baths, or a warm bath, with subsequent sweating; local bleeding and blisters behind the ear may also be resorted to. These measures are, however, only serviceable during the first few days. The galvanic current may also be recommended for the neuritis, although the doubtful results obtained by its application in the severe forms is rather opposed to the probability of its producing any good effects. The stabile application of the anode to the mastoid process of the affected side —the cathode being placed on the opposite side—is useful in the early periods of the disease; subsequently the cathode and anode may be alternately applied on the diseased side.

Too much must not be expected from the use of medicine. The wine of colchicum is of little service; but large doses of iodide of potassium, given for six or eight days in the early stages, have appeared to me to materially shorten the course of the affection in some severe cases. The existence of intermediate forms greatly increases the difficulty of arriving at a correct conclusion in regard to the value of remedies.

Amongst the *direct* means of treatment, electricity is the only one that can be recommended, though even it does not fulfil all that has been expected from it. The galvanic current, which has been so strongly recommended in the reaction of degeneration, has undoubtedly been overestimated. I have shown, in a critical account of the facts hitherto known,[1] that even when the galvanic treatment has been early resorted to, it has not materially shortened the duration of severe forms of rheumatic facial paralysis, and that these recover as rapidly and perfectly with faradic as with galvanic treatment. There is no evidence, therefore, in favor of the supposed superiority of the galvanic over the faradic treatment in this disease.

The results of observation, however, appear to show that, in the slight forms of rheumatic paralysis, each application of electricity is usually followed by an improvement of motility; that in the intermediate form, the daily application of electricity produces a distinct though less marked improvement; and that, lastly, in the severe forms, at a time when the conductivity of the nerves is re-established, the improvement in the motility is materially accelerated by electricity. In all these cases, therefore, electricity should be employed.

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[1] Archiv für klinische Medicin, Band V. p. 88.

In regard to the methods in which it should be employed, I must refer to the observations made in the general part. In the slighter forms it is sufficient to apply it every day or two, or three times a week, for two or three minutes, in the form of a moderately strong faradic current, or of the labile action of the cathode, the anode being placed behind the ear. In the intermediate forms the same treatment is indicated, and in addition the above-mentioned galvanization through the mastoid processes. In severe cases the principal reliance must be placed upon the latter proceeding (it is doubtful whether coincident treatment of the cervical sympathetic can promote the cure), and, generally speaking, the galvanic treatment is to be preferred. (It must not be forgotten, however, that Duchenne obtained good results with the faradic current.) During the first weeks the galvanic treatment may be applied to the periphery of the nerves, and to the muscles, about once a week; but as soon as the traces of motility are observed, this method of applying peripheral irritation may become more prominent, and should be regularly practised. The orbicularis palpebrarum requires special attention, on account of the secondary excitation of the conjunctiva; it is sometimes possible, by the energetic galvanization of the muscle, to increase its tone to some extent, and thus to lessen the amount and frequency of the injurious influences. The strength of current that should be applied must be regulated by the sensibility of the patient and the excitability of the muscles; the treatment may, in general, be commenced with from six to ten of Stöhrer's elements. If the faradic current be used, its strength must be determined by the sensibility of the skin of the face, since the nerves and muscles do not react to it.

In all the other forms of facial paralysis electrical treatment, based on the above-mentioned principles, is indicated, as soon as the primary cause has been removed; and in most cases, when the cause is unknown or the prognosis is doubtful, it is justifiable to try the effects of electricity.

When electricity proves useless, little or no benefit will be obtained from the subcutaneous injection of strychnia, from stimulating liniments, or from cold or warm douches.

Little can be done to relieve secondary contractures. If complete recovery takes place, they disappear of themselves. Their disappearance may be hastened by mechanical extension of the rigid muscles (wooden balls in the cheeks, traction with the fingers, etc.), or by faradisation of the healthy antagonists (Brenner), perhaps also by catalytic action of the galvanic current (stabile application of the cathode) on contracted muscles.

Many methods of operative procedure have been suggested in incurable cases in order to remove distortions (subcutaneous section of antagonists, of the levator palpebræ superioris, operation for ectropion, etc.) Too much must not, however, be expected from these proceedings.

d. *Paralysis of the Muscles supplied by the Nervus Accessorius Willisii.*

A. Eulenburg, l. c. p. 561.—*Duchenne*, Elccris, local. 2. u. 3. Aufl.—*Erb*, Paralyse u. Atrophie sämmtl. vom N. acccss. sin. versorgt. Muskeln. Arch. f. klin. Med. IV. p. 246. 1868.—*Seeligmüller*, Lähmung des Accessor. Willisii. Arch. f. Psych. u. Nerv. III. p. 433. 1871.

This nerve is chiefly distributed to the sterno-cleido-mastoid and trapezius muscles, which, however, receive other branches from the cervical plexus. Its internal branch, which soon unites with the vagus, innervates the muscles of the larynx, part of the velum palati, and some of the pharyngeal muscles.

Etiology.—The causes of this paralysis, which is on the whole of rare occurrence, are probably in part rheumatic and then for the most part affect only the external branch, or one of its twigs, or they may be traumatic, and caused by cuts, stabs, or gunshot wounds in the neck, etc. Paralysis of the abovenamed muscles may also result from compression of the nerve, however produced, whether by disease of the bones of the skull, fractures of the cervical vertebræ, tumors, swelling of the lymphatic glands, and abscesses, as well as from neuritis and neuromata. Lastly, progressive atrophy of these muscles, especially of the trapezii, not unfrequently leads to their complete paralysis.

Symptoms.—We shall here only describe the symptoms of paralysis of the two large muscles supplied by the accessorius. Each of these may be separately attacked, but they may be also coincidently paralyzed; and again each or both may be affected by unilateral or bilateral paralysis.

In *unilateral paralysis of the sterno-cleido-mastoid*, the head is held in a slightly oblique position, in consequence of the unopposed action of the healthy muscle on the other side; the chin is turned towards the affected side, and somewhat elevated, and the head cannot be easily rotated voluntarily in the opposite direction, whilst passive movement can be easily performed. This unnatural position is, however, not very strongly marked, since the rotation of the head can be effected by other muscles. The absence of the well-marked prominence of the sterno-cleido-mastoid, which should appear when the movement, usually effected by this muscle, is resisted, is very characteristic, so that if the chin be supported with the hand, and the patient be directed to move it forcibly downwards or laterally, the muscular belly on the sound side alone projects. When unilateral paralysis has lasted for a long time, contracture of the healthy muscle occurs, and causes persistent obliquity of the position of the head.

With bilateral paralysis of the sterno-cleido-mastoid, the head is held straight, but the corresponding movements, and especially rotation of the head with the chin somewhat raised, can only be performed with difficulty, and with the aid of other muscles. Here, also, the absence of the firm prominence of the muscles in these movements is very characteristic. If the muscles are coincidently atrophied, a slight and otherwise absent depression between the mastoid process and sternum is produced; the neck appears somewhat leaner.

In paralysis of the *trapezius*, the deformity is chiefly expressed by the position of the scapula; this appears to be drawn bodily somewhat downwards and forwards; its inner border is separated from the vertebral column, and placed obliquely, whilst the inferior angle remains relatively nearer to the vertebral column. The acromion process falls

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downwards and forwards, partly owing to the weight of the arms, partly in consequence of the antagonistic action of the rhomboideus and levator anguli scapulæ; in consequence of this the clavicle projects, the supra-clavicular fossa appears deeper than natural, and the posterior and superior angle of the scapula can be felt at its upper border with unusual distinctness.

Partial paralyses of the muscle are not uncommon, on account of its nervous supply being derived from various sources, and these are especially liable to occur in the course of progressive muscular atrophy. The position of the scapula differs to some extent, according to whether the upper, middle, or lower third of the muscle is paralyzed.

Voluntary elevation of the shoulder is limited, or at least is effected in another direction, and by other muscles than normal. The levator anguli scapulæ here acts vicariously, and usually undergoes considerable development in those affected with paralysis of the trapezius, so that it may easily be mistaken for the anterior border of a strongly contracting trapezius. The distinction between these two, however, is not difficult, if a little care be exercised, for the levator chiefly raises the internal and superior angle of the scapula; the belly of the muscle can only be followed to this point; it considerably deepens the supraclavicular fossa, and makes the clavicle stand noticeably off from the thorax; the trapezius, on the other hand, chiefly elevates the acromial end of the scapula, drawing the whole scapula towards the vertebral column, and the belly of the muscle may be distinctly followed to the acromial end of the clavicle. This may also be very distinctly demonstrated by faradic investigation. Moreover, the voluntary approximation of the scapula to the vertebral column (retraction of the shoulder) is considerably interfered with; so far as it is practicable, it is effected by distinctly visible contractions of the rhomboids, which draw the scapula at the same time upwards, and render its border somewhat oblique. Lastly, the elevation of the arm above the horizontal line, notwithstanding the normal action of the deltoid and serratus magnus, is considerably disturbed, because the traction exercised upon the acromion by the upper third of trapezius upwards and backwards is absent; paralysis of the trapezius thus offers, in the abnormal position of the shoulder, and in the disturbances of motion that result, a very characteristic picture.

If both trapezii are paralyzed, these anomalies are present on both sides, the back appears to be more strongly arched in a horizontal direction, because both shoulder-blades have fallen outwards and forwards ; the position of the head is not essentially altered, but some difficulty is experienced in maintaining it in an upright and straight position, the head easily sinking on the chest.

If both the muscles supplied by the accessorius are paralyzed, the symptoms of the double paralysis are combined. In such cases, especially when there is paralysis of the whole accessorius, symptoms are not uncommonly present which result from paralysis of the internal branch : hoarseness caused by paralysis of the laryngeal muscles of the affected side ; nasal intonation of voice, etc., owing to paresis of the velum palati ; and difficulty of swallowing from paresis of the constrictors of the same side. Seeligmueller has observed increased frequency of the pulse in bilateral paralysis, though this is not common in unilateral paralysis (Erb).

There is little to be said in regard to the *electrical relations*. In one case of traumatic paralysis, caused by a stab with a knife in the neck, I found the reaction of degeneration. This would depend entirely upon the cause of paralysis, and upon any secondary trophic disturbances that might be present in the nerves and muscles.

The *diagnosis* is self-evident from the above description. The anomalies of the position maintained and of the motility must be carefully examined ; no mistake should be made in regard to the levator anguli scapulæ, and the movements of the head and shoulder-blade should be investigated with the greatest possible care, accurate comparisons being made with those of the opposite side ; and for this purpose faradic excitation is extremely useful. The preservation of passive motility prevents this from being mistaken for contractures of the antagonistic muscles.

In regard to the determination of the *seat* of the cause of the paralysis, the implication of the several branches, and especially of the branches of the internal division, is of importance. The persistence of contractions of the sterno-cleido-mastoid and trapezius during forced inspiration, whilst the muscles cannot be brought into play by voluntary effort, is very characteristic of centric paralyses, that is to say, of paralyses which have their seat of origin above the pons.

The *prognosis* is determined by the cause of the disease and by the amount of trophic disturbance that may be present.

Treatment.—In addition to the causal treatment, the application of electricity is of primary importance, and should be undertaken according to general principles. In chronic cases, with secondary contractures, appropriate orthopædic treatment is requisite (active and passive gymnastics, tenotomy, supporting apparatus).

e. *Paralysis of the Muscles supplied by the Hypoglossal Nerve.—Glossoplegia.—Paralysis of the Tongue.*

Romberg, l. c. 2 Aufl., I., 3. p. 78, 1851,—*A. Eulenburg*, l. c. p. 546.—*Weir Mitchell*, Injuries of Nerves, p. 335.—See also the Literature of Progressive Bulbar Paralysis.

The hypoglossal nerve, it is well known, innervates all the lingual and most of the muscles attached to the hyoid bone. Its paralysis is consequently indicated essentially by disturbances of the important and extremely delicate motility of the tongue.

Etiology.—Lingual paralysis is not unfrequently a subordinate symptom of various centric diseases. It very rarely occurs as an isolated affection. The tongue is often implicated in hemiplegia from hemorrhage, embolisms, etc. in the central ganglia and cerebral hemispheres. Paralysis of the tongue is a frequent symptom in diseases of the medulla oblongata, in progressive bulbar paralysis, in tabes dorsalis, when it is sufficiently advanced, and in progressive muscular atrophy, if the nucleus and origin of the hypoglossal are implicated in the lesion. Injuries of the spinal cord at a high level, as, for example, in fracture of the atlas, may cause lesion of the hypoglossal nerve. This nerve may also be peripherically injured by diseases of the basis cranii, tumors, enlargement of lymphatic glands, wounds, operations in the neck, etc. As the movements of the tongue ministering to articulation probably possess centres of innervation common to both nerves, it is obvious that in central paralyses both hypoglossal nerves are not unfrequently affected.

Symptoms.—When the tongue is paralyzed on one side, it assumes an oblique position ; on being thrust out, the tip is directed to the paralyzed side, in consequence of the unilateral action of the healthy genioglossus, which pushes the tongue, when protruded, to the opposite side ; the unilateral paralysis of the elevators of the hyoid bone has but little influence in this respect. Isolated contractions of lingual fasciculi of muscle are not unfrequently observed on the sound side, in consequence of which the apex of the tongue can be curved towards the sound side ; or, when efforts to innervate are made, the sound side becomes more prominent. Paresis or paralysis of one half of the tongue, as a rule, is only distinctly observable in the performance of the various movements. When it is at rest on the floor of the mouth, no well-marked difference can be noticed.

When the paralysis is bilateral and complete, the tongue lies immovable on the floor of the oral cavity, appears relaxed and atrophied, not unfrequently presents a constant trembling movement (in consequence of fibrillar muscular contractions), with small wrinkles on its surface. If there be only paresis, the tongue can be protruded, though with some difficulty, and is at once tremblingly retracted ; the performance of the several voluntary movements is accomplished only slowly and incompletely.

The most important disturbances that occur in all forms of glossoplegia are those of *mastication* and of *speech* (masticatory and articulatory glossoplegia of Romberg) ; mastication is interfered with because the masses of food are no longer properly rolled about in the oral cavity, and placed between the teeth ; swallowing is rendered more difficult because the tongue is incapable of directing the food backwards into the pharynx and shutting off the oral cavity from the pharyngeal cavity ; the food consequently remains on the dorsum of the tongue, or lies on its paralyzed half ; food and fluids regurgitate into the oral cavity during deglutition; salivation and swallowing of the saliva is rendered difficult, and the patient is annoyed by being frequently obliged to expectorate.

Disturbance of *speech* may present itself in various degrees of intensity. When the paralysis is unilateral and partial, only those sounds, the pronunciation of which is effected by the tongue, are rendered difficult, and are indistinctly and incompletely articulated; these letters are *s*, *sch*, *l*, *e*, *i*, and at a subsequent period, *k*, *g*, *r*, etc. When the paralysis is bilateral and the tongue has undergone atrophy, speech becomes exceedingly indistinct, muttering, and inarticulate, so that the patient can only express himself in sounds that are scarcely intelligible. (This must be clearly distinguished, on the one hand, from stuttering, which is a form of spasm, and disturbance of co-ordination of the muscles of the tongue; and, on the other hand, from dumbness and aphonia, which are essentially different from the "articular glossoplegia.") Singing, and especially the singing of falsetto notes, is rendered difficult even in the less marked degrees of lingual paralysis.

The diagnosis of lingual paralysis is easily made, since even slight disturbances of the extremely fine and complex motility of the tongue are rendered very evident by the remarkable functional disturbances that result from them. It is seldom that it is requisite to pay great attention to the recognition of finer disturbances. In more complicated cases, as for example those of bulbar paralysis, the disturbances arising from paralysis of the lips or of the velum palati must be distinguished from those caused by the glossoplegia.

The prognosis is entirely dependent on the primary disease, and is hence, as a rule, unfavorable; even in apoplectic hemiplegiæ, in which the motility of other cerebral nerves ordinarily returns quickly, disturbances of articulation and the oblique position of the tongue often persist for a long time. Peripheral paralyses of the hypoglossal nerve usually offer a better prognosis.

Treatment.—The treatment has in but a few recorded cases proved favorable, which might be expected, since there is usually some severe central lesion; at the same time these diseases require direct treatment, and any evident cause of the disease should if possible be removed. In regard to the direct treatment, electricity should be exclusively recommended. Faradisation or galvanization of the tongue may be applied directly from the oral cavity, or by excitation of the hypoglossal nerve in the neck, immediately above the great cornu of the hyoid bone. The primary affection may also in many instances be subjected to and greatly improved by galvanic treatment.

f. *Paralysis of the Muscles supplied by the Cervical and Dorsal Nerves.*

In a practical point of view, it is not very easy to draw a sharp line of distinction between the nerves or plexuses affected in the extremely numerous forms of paralysis that come into consideration under this head, because even under normal conditions there are no well-defined limits, but all imaginable combinations occasionally occur. In order to avoid frequent repetition, especially in regard to the etiology and treatment of these paralyses, we prefer to give a rather general account of them, and shall subsequently treat with more fulness the special symptomatology and the more frequent and important types that present themselves, as, for example, paralysis of the serratus, radial paralysis, etc. Reference to numerous muscles and groups of muscles, which are seldom affected and in which the existence of paralysis is diffcult to recognize, as in the deep muscles of the throat, neck, and back, will be entirely omitted.

A natural classification of the very abundant material may be made by first discussing paralysis of single muscles or groups of muscles in the throat and neck, in the shoulder, back, and abdomen; then the paralyses of the inspiratory muscles; and finally concluding with an account of the isolated and combined forms of paralysis in the regions supplied by the various nerves of the upper extremities.

For detailed information we must refer to the following works:

C. Bell, l. c. p. 324 et seq. 1832.—*Duchenne*, Électris. local.—*A. Eulenburg*, l. c. pp. 565—595.—*Schmidt*, z. Casuist. periph. traumat. Paralysen. Diss. Berlin, 1870.—*Ferréol-Reuillet*, Etudes sur les paral. du membre supér. liées aux fract. de l'humerus. Thèse, Paris, 1869.—*Bernhardt*, Armlähmung nach Schulterluxation. Berl. klin. Woch. 1871, No. 5.—*Seeligmüller*, über Sympathicusaffectionen bei Verletzungen des Plex. brach. Berl. klin. Woch. 1870, No 26.—See, in addition, the various treatises on nervous diseases and electrotherapeutics, the literature of cerebral paralyses, etc.

Paralysis of the Serratus. *Velpeau.* Anatom. chir. 1825.—*Desnos*, Diss. 1845.—*Hecker*, Erfahr. im Gebiet der Chir. u. Augenheilk. 1845.—*Cejka*, Prag. Vierteljahrschrift, 1850.—*Neuschler*, Arch. d. Heilk. 1862.—*Busch*, Langenbeck's Arch. IV. 1. 1863.—*Wiesner*, Arch. f. klin. Med. V. p. 95, 1868.—*Eulenburg* sen., Berl. klin. Woch. 1869, No. 42.—*Caspari*, Arch. f. klin. Med. V. 1868.—*Chvostek*, österr. Zeitschr. f. pr. Heilk. 1871, Nos. 13—16.—*O. Berger*, Lähmung des N. thorac. long. Breslau, 1873 (complete and exhaustive account).

Paralysis of the Muscles of the Back. *Zuradelli*, delle paralisi dei muscoli della Spina. Gazz. med. it. Lomb. 1859, Nos. 18—21.

Paralysis of the Inspiratory Muscles and Diaphragm. *Bell*, l. c.—*Romberg*, l. c. 2. Aufl. I. 3. p. 104.—*Oppolzer*, Lähmung des Zwerchfells. Spitalzeit. 1862, No. 24.—*Duchenne*, Electr. local. II. éd. p. 718.

Paralysis of the Musculo-Spiral (Radialis, Henle). *Zuradelli*, Gazz. med. ital. Lomb. 1858, Nos. 44—47.—*Bachon*, de la Paral. du nerf radial, Paral. des Porteurs d'Eau de Rennes. Mém. de Méd. et Chir. milit. Avril, 1864.—*Brenner*, traumat. Lähmung der Mm. extens. carpi, etc. Petersb. med. Zeitschr. XI. 1866.—Electroth. Unters. II. 1869.—*Billroth*, Krückenlähmung des N. radialis. Wien. med. Woch. 1867, No. 69.—*Gubler*, *Nicaise*, de la tumeur dorsale de la main dans la paralys. saturn. etc. Union méd. 1868, Nos. 78—80, 105. Gaz. méd. de Par. 1868, Nos. 20, 21.—*Laferon*, Rech. sur les paralys. etc. résult. de l'usage des béquilles. Thèse, Paris, 1868.—*Hitzig*, Studien über Bleivergiftung. Berlin, 1868.—*Bärwinkel*, Schmidt's Jahrb. Band 139, p. 118.—*A. Eulenburg*, Bleilähmung. Berl. klin. Woch. 1868, No. 6.—*W. Erb*, Bleilähmung. Arch. f. klin. Med. IV. p. 242, 1868.—*Fournié*, Tuméfact. de la main etc. Union méd. 1869, No. 17.—*Daviot*, über denselben Gegenstand. Thèse, Paris, 1869.—*Webber*, Paralysis of the Radialis, Bost. med. Journ. 1871.—*v. Krafft-Ebing*, Krückenlähmung. Arch. f. klin. Med. IX. p. 125. 1872.—*Busch*, Heilung einer Radialislähmung nach Oberarmbruch. Berl. klin. Woch. 1872, No. 34.—*Savory*, Excis. of the musculo-spiral nerve. Lancet. 1868, Aug. 1.—*Langenmayr*, Casuistik der Radialislähm. Diss. Berlin, 1872.—*Bernhardt*, Differentialdiagnose der Radialisparalyse. Virch. Arch. 54, 1872.—*Panas*, de la paralysie reputée rheumatism. du nerf radial. Arch. génér. Juin, 1872.—*Gombault*, Contribut. à l'histoire anatom. de l'atroph. musc. saturn. Arch. de physiol. 1873.—*Bernhardt*, zur Pathol. d. Radialisparal. Arch. f. Psych. u. Nerv. IV. 3. 1874.—*C. Westphal*, über eine Veränderung d. N. rad. bei Bleilähmung, ib. IV. 3. p. 776.

Paralysis of the Median. *Richet*, Section du nerf médian. Conservat. de la sensibilité de la main. Union méd. u. Gaz. des hôp. 1867. (See also *Duchenne*, El. loc. III. éd.)

1. *Paralysis of Special Muscles and Groups of Muscles of the Neck and Trunk.* (*Neck, Back, Shoulder, Breast, and Belly.*)

Nearly all the muscles that we are about to mention may be separately and independently paralyzed, though it is more common for several to be coincidently affected. The general characters of the disease may in consequence be very complicated, and in many instances great attention is required to recognize the particular muscle the function of which is abolished. We are much indebted to Duchenne for making material advances in our means of diagnosis.

Paralysis of the pectoralis major and pectoralis minor.—These muscles, which are supplied by the anterior thoracic nerves (Henle), are very rarely separately paralyzed, and their paralysis is usually only a subordinate symptom of more complex forms of paralysis (traumatic paralysis of the brachial plexus, hemiplegia, and not unfrequently progressive muscular atrophy). It is recognized at once by the impairment or total loss of the power of adducting the arm to the thorax, and by the inability of the patient to seize the opposite shoulder with the hand, or to offer any resistance to passive abduction of the arm. If atrophy be also present, the subclavicular fossa is considerably deepened, the ribs and intercostal spaces are strongly marked, and may be distinguished with unusual facility, and the anterior wall of the axilla is reduced to a flaccid fold of skin.

Paralysis of the rhomboidei and levator anguli scapulæ, which are supplied by the nervus dorsalis scapulæ (Henle), is recognizable with great difficulty, since it does not occasion any material alteration in the position of the scapula when the arm is at rest. Forced elevation of the shoulder is rendered impossible. It is only when there is complete paralysis and atrophy of the trapezius that the paralysis of the rhomboidei can be easily diagnosticated by the total incapacity of the patient to draw the scapula towards the vertebral column, while paralysis of the levator anguli scapulæ is recognized by the failure to effect the characteristic elevation of the scapula.

Paralysis of the latissimus dorsi is also an affection of extreme rarity. (The muscle is supplied by the subscapular nerves.) It is most commonly seen as a subordinate symptom of progressive muscular atrophy. The principal resulting disturbance is that the arm cannot be adducted with the usual amount of force, and that the ability to bring the raised arm forcibly downwards is diminished or abolished. The arm and shoulder cannot be drawn forcibly downward, nor the hand be brought with the usual facility to the buttock. Rotation of the arm is but little interfered with, since this movement can be effected by the muscles. When the body is at rest, and the arm is allowed to hang by the side, little or no deformity is observable.

Paralysis of the inward and outward rotators of the upper arm not unfrequently occurs in connection with paralyses of other adjoining muscles, and then exhibits tolerably characteristic symptoms, which however are usually only discoverable on close investigation.

If the outward rotators (the *infraspinatus muscle*, which is supplied by the suprascapular nerves, and the *teres minor*, supplied by the axillary) be paralyzed, considerable disturbances are experienced in many occupations in which the rotation outwards of the arm is required. As Duchenne has shown, this occurs in writing and drawing, in which the formation of straight lines from left to right is in part dependent upon the contraction of the infraspinatus and teres, which movements are rendered difficult or impossible when these muscles are paralyzed. The same occurs in all occupations requiring the use of the needle, as the above-named muscles are brought into play every time the thread is drawn out. If the arm be rotated inwards, it cannot be voluntarily rotated outwards, as is very well shown by the rectangularly bent forearm, which acts as a pointer. The increased depression of the infraspinatus fossa, present in so many instances, serves to establish the diagnosis.

Paralysis of the inward rotators (the *subscapularis*, *the teres major*, and in part also the *latissimus dorsi*), which are all innervated by the subscapular nerves, occasions still more remarkable disturbance, as all movements with the hand towards the opposite side of the body or head are rendered difficult or impossible. When the arm is rotated outwards, it cannot be voluntarily rotated inwards. On account of the preponderance of the action of the outward rotators, the arm is constantly maintained in a state of abnormal rotation outwards, and the function of the outward rotators is thus materially impaired. These paralyses are, however, easily distinguished from contractures by the fact that passive motility is preserved. Their etiology, the electrical relations, and the treatment are nearly the same as in paralyses of the serratus.

Paralysis of the *musculus serratus anticus major* belongs to the more frequent and important paralyses of isolated muscles. It has long excited the interest of physiologists and pathologists, and an extensive literature exists in regard to it. On the whole, however, the relations of this form of paralysis are tolerably simple, and are now perfectly understood. (See especially the exhaustive work of O. Berger.)

The muscle is innervated by the nervus thoracicus posterior (Henle), or longus, and receives no other motor branches. The exposed position of this nerve, rendering it liable to many injuries, as well as the length of its course, sufficiently explains the *etiology* of paralysis of the serratus. In the great majority of cases the paralysis is peripheral, and arises from *direct injury* to the nerve, such, for instance, as may result from carrying heavy loads on the shoulder (Wiesner), from pressure or contusion, blows, and concussion of the shoulder, gunshot wounds; but it is doubtful whether mechanical compression and injury experienced by the thoracicus longus, as it perforates the scalenus medius, when violent and repeated movements of the shoulder are performed, can be the cause of paralysis, though it is no doubt highly probable. At any rate, paralysis of the serratus, both unilateral and bilateral, has not unfrequently been observed after *over-exertion of the muscles of the shoulder*, as in mowers, puddlers, shoe- and rope-makers. These various circumstances explain why paralysis of the serratus muscle is far more frequent in men than in women, and chiefly occurs on the right side. "Catching cold" is also a frequent cause, numerous cases having been reported in which the affection has been produced by exposure to draughts, by sleeping on damp ground or near a damp wall. Finally, some cases of this form of paralysis have been observed after typhoid fever.

Paralysis of the serratus is very seldom a symptom of spinal or cerebral paralysis, and is then only one amongst many minor paralytic phenomena. It much more frequently forms a symptom of *progressive muscular atrophy*, especially of that characteristic form which begins in the muscles of the back and shoulder blade. The serratus is rarely first affected, becoming independently atrophic and paralyzed. Far more frequently other muscles are coincidently affected, especially the trapezius (the lower portion of which, according to Duchenne, is almost always paralyzed coincidently with the serratus), the latissimus dorsi, the rhomboidei, etc. The occurrence of bilateral paralysis and atrophy is of course not a rare event.

The *symptoms* of paralysis of the serratus are so characteristic that it is difficult to misinterpret them. They are often preceded for some time by neuralgic pains in the region of the supraclavicular branches of the brachial plexus. With this is associated increased difficulty in the performance of certain movements, which first leads the patient to seek for medical advice. All the symptoms that are at this time present may be sufficiently explained by the physiological action of the muscles (so fully discussed by O. Berger), which influences, on the one hand, the position of the scapula when at rest and during the performance of certain movements, and, on the other hand, the vertical elevation of the arm. Thus we find that, whilst at rest with the arm hanging down, the scapula is somewhat raised and approximated to the vertebral column, and so rotated upon its axis that its inferior angle is approximated to the vertebral column, its anterior border inclined downwards, and consequently its inner border directed somewhat obliquely upwards and outwards; hence we observe a slight wing-like standing off of the inner border, and especially of the inferior angle of the scapula from the wall of the thorax. All these symptoms are caused by the preponderance, when the arm is at rest, of the antagonistic muscles (rhomboidei, levator scapuli and trapezius) over the paralyzed serratus. If these muscles are also paralyzed, the deformity is less marked. Much more striking disturbances immediately make their appearance when certain movements are performed; thus when the patient is directed to raise the extended arm, it is seen at once *that he cannot raise it above the horizontal level*, because the rotation of the scapula forwards and the elevation of its anterior angle, which is effected by the serratus, is no longer accomplished, and without this it is impossible to raise the arm to the vertical position.[1] If this movement be replaced by forcible fixation and rotation of the scapula forwards, the elevation of the arm can be immediately performed. If the arm be raised horizontally in the plane of the body, the inner border of the scapula, instead of being removed farther away from the vertebral column, passes more and more inwards towards the column, pushing a considerable mass of muscle before it; if there be bilateral paralysis, the inner borders of the scapulæ may actually touch one another. If the raised arm be brought forward, the inner border of the scapula becomes more and more separated from the wall of the chest, standing off in a wing-like manner, and to such an extent that a deep fossa is formed in which the hand may be easily laid, so that the inner surface can be felt; whereas under normal conditions the inner border of the scapula is firmly applied to the wall of the thorax in this movement. In bilateral paralysis the scapulæ thus inclose a deep hollow, in which the muscular bellies of the rhomboidei distinctly project. By this symptom alone—the inner border of the scapula projecting in a wing-like manner when the arm is raised in a forward direction—paralysis of the serratus may be recognized at once with certainty. If contraction of the muscle can be effected by faradic irritation of its nerves, the characteristic deformity is at once removed.

In addition to the difficulty of raising the arm, which is particularly annoying to the patient, other movements are also interfered with; thus it is difficult to cross the arms in front of the chest, and to perform the movement of the apex of the shoulder forwards, as, for example, in delivering a forward blow in fencing; the patient also offers less resistance on the paralyzed side to forcible retraction of the shoulder, etc.; on the other hand, respiration is not in any way disturbed even when the paralysis is bilateral; the serratus is not, as was believed by Bell, Stromeyer, and others, a muscle of inspiration.

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[1] One of my patients, a young girl, was indeed able to raise the arm to the vertical position, but only by throwing the body backwards and swinging the arms upwards, thus effecting dislocation of the head of the humerus downwards.

As a rule, *disturbances of sensibility* are rarely or only accidentally present, since anæsthesia, neuralgia, and hyperæsthesia may occur in the region of the brachial plexus from the same cause as that which has led to the paralysis of the long thoracic.

Considerable *atrophy* of the affected muscle is usually observed in progressive muscular atrophy, as well as in severe traumatic, neuritic and similar paralyses; this, of course, takes place in all instances, but it is altogether absent or only slight in degree in paralyses of centric origin, or in slight paralyses resulting from rheumatism or from pressure.

The electrical relations are similar in this form of paralysis; that is to say, in traumatic and severe rheumatic forms of paralyses, the reaction of degeneration is present to an extent corresponding with the stage of the paralysis; in paralysis from progressive muscular atrophy there is simple diminution of the electrical excitability corresponding to the degree of atrophy; in paralyses of centric origin, and in those produced by slight pressure, there is either no change or only a slight diminution of the electrical excitability. In most of the cases hitherto observed, the examination has been incomplete and only diminution or loss of faradic excitability has been noted.

In regard to the *course* pursued by paralysis of the serratus muscle, we need only say that, after having made its appearance suddenly or gradually, it may remain stationary for a variable period, often for many months, and it only very gradually terminates in recovery. Rheumatic paralyses and those arising from slight pressure almost always recover. Traumatic paralyses are usually of long duration, and are not unfrequently incurable, and this is still more certainly true of paralyses arising from progressive muscular atrophy. In favorable cases the muscle long remains feeble, and there is a disposition to relapses. In protracted cases gradual contracture of the antagonists takes place, which becomes progressively more marked and materially increases the abnormal conditions produced.

It is not difficult to recognize paralyses of the serratus after the above description, since the *diagnosis* is sufficiently established by the patient's inability to raise the arm to the vertical position (whilst this can easily be effected passively), and by the characteristic displacement of the scapula. The passive motility of the scapula, and the wing-like standing off of the internal border of this bone, prevent the disease from being confounded with contracture of the antagonists. Paralysis of the other muscles attached to the scapula cannot be easily mistaken for paralysis of the serratus, if a little attention be paid to the case.

The *prognosis* of paralysis of the serratus is determined by the cause of the disease, the degree of the atrophy, the alterations of the electrical excitability, and the time that the disease has lasted.

Paralysis of the dorsal muscles.—Under this term we group those muscles upon whose action the movement, fixation, and upright position of the vertebral column depend. It is almost always impossible to define exactly the particular muscles which are affected in these forms of paralysis. We are only tolerably accurately acquainted with paretic and paralytic conditions of the extensors. In youth various degrees of weakness of the dorsal muscles are often present, sometimes on one, sometimes on both sides, and affecting a greater or less extent of the back. They lead to definite forms of spinal curvature with which we have nothing further to do in this place. Rheumatic affections not unfrequently cause paralysis of one or several of the dorsal muscles, and injuries affecting the vertebral column and the back may have the same effect. Paralysis of the dorsal muscles are now and then, but very rarely, observed as a symptom of cerebral paralyses; they are, however, more frequent in spinal disturbances of motility. In paraplegiæ which gradually rise towards the chest or upper extremities, nothing is more common than the symptoms of paralysis of the extensors of the back. Moreover, progressive muscular atrophy not unfrequently extends to the dorsal muscles, and, in the so-called pseudo-hypertrophy of muscles, the weakness of the large extensors of the back in the lumbar region forms a very characteristic feature of the disease. In a few cases I have observed this condition of weakness with atrophy developed in young persons without any obvious cause.

The muscles principally affected are the sacro-lumbalis and the latissimus dorsi with their continuations towards the neck and head, and also the small muscles between the several vertebræ. It is impossible to distinguish the several parts of these paralyzed muscles from one another clinically, and it is sufficient to confine ourselves to the determination whether the paralysis has its seat in the lumbar, dorsal, or cervical portion. If the muscles be paralyzed on both sides, a gradually increasing posterior curvature of the spinal column occurs at the affected part (paralytic kyphosis), which is usually most marked in the dorsal region and is less distinct in the lumbar and cervical regions. When the extensors of the dorsal region are paralyzed, the vertebral column forms a large and equable curve, the patients appear bent and doubled up as in advanced age, and are unable to straighten themselves voluntarily, and to hold themselves upright for any length of time, whilst passive straightening of the vertebral column can be effected with tolerable facility, and distinguishes the affection from kyphosis consequent on muscular contracture, or disease of the vertebræ. If the paresis or paralysis be only unilateral, the various degrees of paralytic scoliosis are produced. There are numerous grades of these paretic conditions leading to manifold varieties in the amount and extent of the consecutive deformity, and in the power of *the patient to rectify* this deformity by voluntary muscular effort.

Paralysis of the *extensors in the lumbar region* presents very characteristic features. The patients at once attract attention on account of the curious position they assume. They bend the upper part of the body backwards, to compensate for the bending in of the lumbar vertebræ, so that the centre of gravity of the upper part of the body falls behind the centre of gravity of the whole body ; the balance is then preserved exclusively by the abdominal muscles, and if the upper part of the body is moved forward, so that its centre of gravity advances too far in that direction, the paralyzed lumbar muscles are no longer capable of holding it back, and it sinks or falls forward; the patient is unable to raise himself again without the aid of his hands, and the mode in which such patients climb with the hands on their legs, by all kinds of movements with the shoulders and arms, raising the upper part of their body, and gradually bringing it so far back that they are able to balance it again with the muscles of the abdomen, is in the highest degree characteristic of the affection. Paralysis of the muscles of the lumbar region may, in fact, be immediately recognized by the deep hollow of the back, the backward inclination of the upper portion of the body, and the head bent somewhat forward in standing and walking. In walking, a remarkable oscillating movement of the trunk is observed from side to side, on account of the absence of the synchronous contraction of the lumbar muscles on the same side, which should accompany the forward movement of the leg, and on account of the regular balancing movement of the body which results when steps are made in a forward direction. On the other hand, when the patient seats himself, the upper part of the body sinks down, and the patient presents a slight kyphosis of the vertebral column in the lumbar region. There is almost always coincident atrophy of the lumbar muscles, the thick muscular bellies of which are greatly diminished in size, whilst the spinous and transverse processes of the vertebræ are unnaturally distinct.

If *the muscles of the neck* are alone paralyzed, it is indicated essentially by the position of the head; this can no longer be carried erect; it sinks forward as soon as the muscles become fatigued, and the centre of gravity is placed too far forwards. The patient, however, can still raise it by a peculiar swinging movement, and then usually carries it inclined backwards, supported only by the anterior muscles of the neck. This position is highly characteristic. The electrical conditions accompanying these forms of paralysis have been as yet but little investigated; it is only possible to test the direct excitability in the affected muscles; and this has, as a rule, been observed to be simply lowered.

Paralysis of the abdominal muscles is extremely rarely met with as an independent affection, which is easily explicable when the great variety of sources from which they receive their supply of nerves is considered ; it is, on the other hand, a very common symptom of extensive centric, and especially of spinal paralysis, and more rarely of progressive muscular atrophy.

The symptomatology is very simple. In unilateral paralysis, as, for example, in hemiplegia, the navel is drawn towards the sound side with each movement of forcible expiration; when the paralysis is bilateral the most remarkable symptom is the weakness of the expiration and of all respiratory reflex acts, such as coughing, expectoration, sneezing, crying, etc.; hence the danger to which paraplegic patients with paralysis of the abdominal muscles are exposed, if they happen to suffer from bronchial catarrh, as they are unable either to cough or to expectorate. Secondly, it may be observed that the power of compressing the abdomen is impaired ; hence the slowness and difficulty of evacuating the contents of the rectum and bladder. The abdomen is large and protuberant, and its walls relaxed ; the well-known reflex acts proceeding from the skin of the abdomen may be either abolished or exalted ; the patient is unable to raise the head and upper part of the body from the recumbent posture or to sit up in bed without the assistance of his hands ; in walking or standing the upper part of the body is bent slightly forwards and balanced exclusively by the lumbar muscles, because each shifting of the centre of gravity backwards renders the patient liable to fall backwards, because the feeble abdominal muscles are incapable of drawing the trunk forwards. This symptom is of course only well-marked when the paralysis is isolated, since if paraplegia be also present, the patient can neither stand nor walk.

In regard to the prognosis of all the paralyses that have just been described, we must refer to what has been stated in the introductory section. The treatment also of these paralyses must be conducted on general principles. After careful investigation and if possible, fulfilment of causal indications, electrical treatment should be at once commenced, providing there are no distinct contra-indications, or the prognosis is not absolutely bad. Either the faradic or the galvanic current may be tried, and we must again refer to the general observations that have been already made. Great care should be taken to obtain an exact and correct localization of the current upon the nerves and muscles, and also to regulate exactly the strength employed. In some instances brilliant success is obtained, whilst in other instances it fails, which is probably dependent on the nature of the primary cause, or the duration of the affection, and on the degree of the trophic disturbances that may be present. For all details we must refer to the treatises on electro-therapeutics.

In many cases, though only where the disease is merely a paresis, or where considerable improvement has already commenced, a rational system of gymnastics practised with energy and perseverance may have a beneficial effect. Sometimes, especially in incurable cases, appropriate supporting and orthopædic apparatuses may be employed to replace the deficient muscular action, but comparatively little can be effected by these means in paralysis of the muscles of the trunk.

Of course in all appropriate cases general tonic treatment must be pursued, with the employment of embrocations, baths, douches, liniments, change of air, etc.

2. *Paralysis of the Muscles of Inspiration.*

The muscles of inspiration are widely separated from one another in point of position, and are supplied by very different nerves. Complete paralysis consequently only occurs when the respiratory centres in the medulla oblongata are damaged, or when the motor nerves proceeding from these centres, and which for the most part run in the lateral columns of the cord, are destroyed. When this occurs upon both sides, as in compression of the spinal cord from fracture of the uppermost cervical vertebræ, rapid death is inevitable. Unilateral paralysis of the respiratory nerves in their course through the spinal cord, which is now and then observed, does not immediately endanger life. The respiratory centres may be paralyzed as a consequence of various degenerative processes taking place in the medulla, as well as from the action of various poisons. The respiratory processes are not in general materially interfered with in cerebral paralyses; muscles paralyzed, so far as the will is concerned, may be observed in hemiplegic patients to react readily to excitations proceeding from respiratory centres.

Peripheral paralyses of the respiratory muscles usually affect only individual muscles. Amongst these may be mentioned the intercostals, the scaleni, and other auxiliary inspiratory muscles, which, however, are all of subordinate importance, since they can cause no material disturbance of the respiratory acts. On the other hand, *paralysis of the diaphragm*, which is the most important muscle of inspiration, always seriously interferes with the respiratory process.

Paralysis of the diaphragm is, upon the whole, of rare occurrence; it may result from inflammation of the serous membranes, investing the diaphragm, as in pleurisy and peritonitis, in which case it results from an extension of the inflammation, hyperæmia, and transudation from the membrane to the muscle. It may also be a symptom of progressive muscular atrophy, in which it almost always first occurs at an advanced stage of the disease, and hastens the fatal issue. It is occasionally observed in hysterical patients. Duchenne has noticed it as a consequence of lead-poisoning. According to Oppolzer, it frequently occurs during adolescence without apparent cause; lastly, "catching cold" has been in many instances assigned as a cause, acting either by producing rheumatic paralysis of the phrenic nerve, or by causing muscular rheumatism of the diaphragm. It is obvious that traumatic paralyses and paralyses from compression of the phrenic nerve in the neck may occur. Diaphragmatic paralysis is usually bilateral, so that the whole diaphragm is paralyzed; it may, however, be unilateral or partial.

The *symptoms* of the affection are highly characteristic; in inspiration the epigastrium and the hypochondria are drawn inwards instead of being curved outwards, as under normal conditions, and this difference is particularly observable when the inspirations are deep and long; if the hand be placed upon the epigastrium during inspiration, the pressure of the descending diaphragm cannot be perceived; in expiration the epigastrium projects. When the paralysis is unilateral, these symptoms occur only on one side, but may still be distinctly detected by palpation.

In these cases, when the patient is at rest, there is only moderate dyspnœa, and the frequency of the inspirations is but slightly increased; but if the smallest exertion be made, or any excitement felt, considerable dyspnœa is experienced, and the frequency of respiration rises to 40 or 50 in a minute. On walking, and especially on ascending stairs or mountains, such patients are compelled to stop frequently to breathe, the voice becomes weaker, and loud and continuous speaking becomes impossible. Strictly speaking, it is incorrect to maintain that there is increased difficulty in performing expiration and the associated reflex acts (coughing, expectoration, and sneezing), since in all these the diaphragm is relaxed; its paralysis, therefore, only affects expiration by causing the preceding inspiration to be incomplete, thus diminishing the quantity of air disposable for the act of respiration. For the same reason, life may be in the greatest danger in intercurrent catarrh, pneumonia, etc., because the power of inspiration is diminished. Furthermore, in consequence of diaphragmatic paralysis, the compressive action of the abdominal muscles is materially interfered with, and this is manifested, especially, by difficulty of defecation.

The *electric excitability* of the phrenic nerves has generally been found to be preserved. A. Eulenburg has, however, found, in one case of rheumatic diaphragmatic paralysis, that it was considerably lowered. Precise information on this point is, however, wanting.

The *diagnosis* is not difficult, after what has been already stated; partial paralyses alone can present any considerable difficulty. The nature of the disease may be determined by the anamnesis and by objective research.

The *prognosis* depends on the cause. In rheumatic and hysterical paralysis it is highly favorable; in saturnine intoxication it is at least very doubtful; whilst in progressive muscular atrophy it is very unfavorable, although even then it is sometimes possible, by early and appropriate treatment, to bring the diaphragm again into use and to preserve its functional activity for some time (Duchenne). There is no direct danger to life from paralysis of the diaphragm itself, though this is common from the accidental complications that may occur ; under all circumstances the activity of persons thus affected is considerably diminished.

The treatment must first be directed to the causal indications and to the removal of rheumatism, hysteria, progressive muscular atrophy, saturnine intoxication, etc. In regard to the direct treatment, little time should be lost in the employment of counter-irritants, irritating liniments, etc., but recourse should early be had to faradisation or galvanization of the phrenics, which can easily be applied in the neck over the scaleni. Irritation is best effected by placing one pole upon the neck whilst the other is placed in the vicinity of the attachments of the diaphragm to the ribs, or, in some instances, on the back of the neck ; the current should be tolerably strong.

3. *Paralysis of the Several Nerves and Muscles of the Upper Extremity.*

The paralyses of the upper extremity are extraordinarily numerous and varied, and complex combinations occur, of which it is scarcely possible to give an exhaustive account ; we shall therefore, in the first instance, describe systematically those paralyses that occur in the region of the chief nerve trunks of the upper extremity, and shall then append a few brief observations in regard to their more ordinary combinations.

a. *Paralysis of the Muscles supplied by the Circumflex (Axillary, Henle) Nerve.*

This nerve supplies the principal part of the deltoid muscle (the anterior part of which, however, receives a few branches from the anterior thoracic nerves), innervates, in addition, the teres minor, and gives sensory branches to the skin of the upper arm. The following causes are those which most frequently lead to its paralysis :—*Injuries* affecting the shoulder and shoulder-joint, or the deltoid muscle itself, by which the circumflex nerve is crushed or torn ; hence, concussions, contusions, blows or falls upon the shoulder, gunshot wounds, and especially dislocations of the head of the humerus backwards, are very frequent causes of deltoid paralysis. *Rheumatism and chronic inflammation of the shoulder-joint* very frequently cause paralysis of the deltoid, partly because neuritic processes extend along the course of the circumflex nerve, and partly in consequence of chronic inflammation (associated with atrophy) taking place in the muscle itself. "Catching cold" is one of the more frequent causes of this paralysis, and it may also be produced by neuritis. Lastly, it is not unfrequently a symptom of paralyses affecting the brachial plexus to a greater or less extent, of central paralyses of all kinds, of saturnine paralyses, and of progressive muscular atrophy.

The symptoms are almost exclusively those of paralysis of the deltoid muscle, since paralysis of the teres minor can scarcely be recognized unless the infraspinatus be also incapable of discharging its function. When the deltoid is paralyzed, the arm cannot be raised; it lies flat and immovable against the wall of the thorax, from which it can scarcely be separated. When attempts are made to raise the arm, the deltoid remains quite relaxed, which distinguishes it from anchylosis of the shoulder-joint; it is also impossible to raise the arm in a forward direction.

The shoulder-joint becomes relaxed and the muscle frequently atrophied, whilst, in severe cases, the joint becomes so loose that a deep groove can be felt through the atrophied deltoid between the head of the humerus and the articular surface of the scapula. It is rare to find any sensory disturbances in the region of distribution of the circumflex nerve, though pain in the shoulder-joint and in the substance of the muscle itself is of common occurrence.

The *electrical excitability* may be, in the first instance, normal, and may then gradually undergo diminution, especially in progressive muscular atrophy, and in the paralysis that results from rheumatism of the shoulder-joint; the various phases of the reaction of degeneration may also be presented. These are often limited to particular parts of the muscle, and often are only incompletely developed, perhaps in consequence of the muscle receiving its supply of nerves from different sources. The electrical excitability rarely remains quite normal.

When the cause of paralysis is persistent, the atrophy gradually becomes more and more marked; the joint becomes loose; in many cases anchylosis of the shoulder-joint takes place, and the arm remains more or less useless. If recovery takes place, the movement of the arm at the shoulder-joint is gradually restored; the patient becomes able to raise the arm to a slight extent, then to the horizontal line, and, finally, to the vertical, providing that the other muscles of the scapula, and especially the serratus anticus, remain intact; coincidently with all this, the atrophy and the alteration of the electrical excitability gradually disappear.

The *diagnosis* of this paralysis, in consequence of the impaired functional activity of the deltoid, is extremely easy. It is only possible to mistake it for anchylosis of the shoulder-joint, from which it can be at once distinguished by the passive motility of the arm and the absence of contraction in the muscle, when attempts are made to innervate it. A little attention only is requisite in order to diagnosticate those cases in which anchylosis and paralysis of the deltoid are coincidently present.

The *prognosis* rests almost entirely on the nature of the primary disease, and may readily be formed on the general principles that have already been mentioned.

b. *Paralysis in the Region of Distribution of the Musculo-cutaneous Nerve.*

The muscles supplied by this nerve are the coraco-brachialis, the biceps, and the brachialis anticus, which last receives an additional branch from the musculo-spiral nerve. Paralysis of this nerve, or of either of the two last-named muscles, always leads to impairment or complete impossibility of flexing the fore-arm on the upper arm. This is particularly well marked when an attempt is made to bend the arm in a position of supination, because then the flexor action of the supinator longus is no longer exerted. The seat of the cause of the paralysis in the nerve may often be ascertained by the presence of anæsthesia in the course of the radial border of the forearm. This paralysis is scarcely ever met with as an isolated affection, but is usually associated with other paralyses in the region of the brachial plexus, and has the same pathogenesis and prognosis.

c. *Paralysis in the Region of Distribution of the Musculo-spiral (Radialis, Henle) Nerve.—Musculo-spiral Paralysis.*

Of all the paralyses occurring in the region of the brachial plexus, that of the musculo-spiral nerve is the most frequent. It may result from many different causes affecting the musculo-spiral nerve and the muscles supplied by it, and it appears to be particularly liable both to peripheral and to central causes of paralysis, so that in cerebral hemiplegiæ the muscles supplied by this nerve are usually most severely affected, whilst of traumatic paralyses and of those arising from chill in the upper extremity, it is by far the most common; and, lastly, saturnine paralysis, affecting the upper extremity, attacks almost exclusively the muscles supplied by it. These circumstances sufficiently explain the voluminous literature that exists in regard to paralysis of the musculo-spiral.

Etiology.—Slight and severe injuries which affect the very exposed musculo-spiral nerve as it winds round the upper arm, or even at a higher point, are undoubtedly among the causes of musculo-spiral paralysis. In fact, the majority of cases usually described as rheumatic are in all probability referable to slight injuries of or moderate pressure exerted upon the nerve in the upper arm. Panas has demonstrated this in a very satisfactory manner, and I can support his views by my own experience. In cases of "sleeping on damp earth," or "near a moist wall," or of "exposure to a draught of air," the true cause of the paralysis may almost always be shown to be compression of the nerve. The seat of the paralysis below the giving off of the branches to the triceps muscle, which always remains intact, is an argument for its mechanical mode of origin.

The various ways in which such injuries may be inflicted are innumerable. Compression of the nerve during a deep and long sleep is undoubtedly extremely common; hence the frequency of this paralysis in those who fall asleep in a certain position on the ground, on steps and benches when intoxicated or in a state of great exhaustion. In such cases, either the arm is compressed by the body against the ground, or the nerve is compressed by the head resting on the arm, as on a pillow; or, lastly, the arm, whilst supporting the head, is compressed at its outer part against the corner of the chair, step, etc. The paralysis appears as soon as, or shortly after, the patient awakens. The etiology of paralyses of the musculo-spiral may be similarly observed, according to Brenner, in Russian coachmen, who fall asleep with the reins wound round the upper arm; or in prisoners, whose upper arms, according to the Russian mode, are bound together behind the back; or in Russian infants, who are tightly swathed with the arm lying in close contact with the body, and are then allowed to sleep for a long time on one side. The same is observed in the water-carriers of Rennes (Bachon), and is due to their carrying heavy pails, filled with water, which they compress with the arms against the chest in such a manner that the handle is firmly applied to the outer surface of the upper arm. Amongst the slighter forms of injury may be mentioned the pressure of improperly-constructed crutches (either too long, badly made and padded, or not properly provided with support for the hand), which compress the musculo-spiral, as well as other nerves of the brachial plexus in their course through the axilla, against the humerus, and thus produce "crutch paralysis."

Many cases of severe traumatism, followed by paralysis of the radial nerve, have been reported, and amongst them may be mentioned blows on the arm, a kick from a cow, cuts, stabs, and gunshot wounds, fracture of the humerus and abnormal formation of callus (Busch, Ollier and Ferréol-Reuillet), dislocation of the shoulder, etc.

The frequency of rheumatic paralyses of the musculo-spiral nerve, originating in various ways from exposure to cold, is less than that of paralyses from injury. In future it will be requisite to make a careful examination of the causes, to determine whether rheumatic paralyses of the musculo-spiral really occur; they are certainly by no means so frequent as has hitherto been believed.

To the same category belong paralyses of the musculo-spiral, arising from neuritis, of which Bernhardt has recently published a case, and of which several have also fallen under my own observation.

Hysterical paralyses of the musculo-spiral are of very rare occurrence, whilst the implication of the muscles supplied by this nerve, in central and especially in cerebral paralyses, is very common.

Lastly, lead-poisoning must be mentioned as one of the most frequent causes of paralysis of the musculo-spiral. It generally first makes its appearance in the later stages of the poisoning, having been preceded by colic, arthralgia, and icterus, whilst it rarely constitutes a primary symptom of the poisoning. The paralysis commences, almost without exception, in the muscles supplied by the musculo-spiral in the forearm, and especially the extensor communis digitorum, subsequently extending to the others, and not unfrequently affecting ultimately the hand, upper arm, and shoulder, as well as the muscles of the lower extremity. A full description of lead paralysis will be given in another part of this work; but we shall here give a short account of the symptoms of lead paralysis in the musculo-spiral region, partly on account of its diagnostic importance, and partly on account of its high scientific interest.

This is not the place to enter into any full details in regard to the still obscure *pathogenesis of lead paralysis.* The question will probably soon be solved, since very recent investigations have furnished a series of important results in regard to the anatomical alterations produced, though they have not yet been able to determine the seat of the paralysis. (See especially the works of Lancéreaux, Gombault, Bernhardt, Westphal.) It may, however, not be out of place to mention, briefly, the various opinions that are held on this point, and to indicate the direction in which future researches should be made. It cannot be doubted that the seat of paralysis should not be sought for in the muscles. In addition to the reaction of degeneration, which is so frequently present (see below), and the existence of paralysis, before advanced atrophy has occurred, the analyses of Heubel and Bernhardt are decisively opposed to this view. The same arguments oppose the attempts made to explain the symptoms by Hitzig, who attributes lead paralysis to a venous stasis on the extensor side of the forearm, and by Bärwinkel, who regards arterial ischæmia of the same parts as the cause, even if it should be admitted that these disturbances of circulation could act directly or by increased deposition of lead in the muscles. Moreover, these views are wholly insufficient to explain the extension of the paralysis to other muscular regions, as the deltoid, interossei, lower extremities, etc. Scarcely a doubt can exist, then, that we have here *a primary lesion of the nervous system*, either affecting the trunk of the musculo-spiral nerve (thus constituting a peripheral paralysis) or involving the nerve at some point in the central organ, and most probably in the spinal cord (thus constituting a central paralysis); these two possibilities are still under discussion.

The presence of well-marked reaction of degeneration appeared at first to be in favor of a peripheral lesion of the nerve trunks in many cases of lead paralysis, because it was formerly believed that this *only* occurred in peripheral paralyses; but since its discovery in the spinal paralysis of children, this view is either incorrect or at least very uncertain, and cannot be regarded as demonstrating the peripheral seat of the paralysis.

The anatomical characters presented by the muscles and nerves are undoubtedly in favor of the view that the paralysis is peripheral. Lancéreaux, Gombault, and Bernhardt found that the atrophied, grayish-red or whitish, tough muscles presented indistinct, transverse striation, multiplication of nuclei, and hypertrophy of the interstitial connective tissue—just those changes which I in due time pointed out in peripheral traumatic paralyses, and which are also seen in progressive muscular atrophy. Gombault observed a high degree of degeneration and atrophy, fatty granular degeneration, abundant interstitial hypertrophy of connective tissue, with multiplication of nuclei; but states, on the other hand, that the cylinder axes are preserved intact, with abundant multiplication of the nuclei of the nerves. (Coincidently with this, the nerve roots and spinal cord remain unaffected.) Lancéreaux also describes granular fatty degeneration of the medulla with disappearance, in places, of the nerve fibres; lastly, Westphal has lately made a very interesting discovery in a case of lead paralysis of about two years' duration. He found, namely, that in the musculo-spiral nerve, besides a few normal fibres, there were numerous fasciculi of fine, obviously regenerated nerve fibres, quite analogous to those that were originally described by Remak as occurring in the later stages of regeneration, after section, and which have been recently again described by Neuman and Eichhorst. But where regeneration is present, there degeneration must previously have occurred; and we may therefore admit that in the earlier stages of the paralysis, degenerative processes occur in the nerves, and these are subsequently replaced by regenerative processes. Unfortunately there is no proof in the case quoted by Westphal of a peripheral cause of the degeneration; there was no known compressing cause, nor any neuritis; even the interstitial hypertrophy of connective tissue, which is constant in peripheral traumatic paralyses, was lacking, and—which is very noticeable—in every partially altered nerve fasciculus there were also numerous well-preserved fibres; this hardly coincides with the idea of a peripheral cause of paralysis affecting the *nerve trunk*. The nerve roots have not as yet been sufficiently investigated. No anomalies of importance have been observed in the spinal cord under the microscope, although microscopically the right anterior horn has been noticed to be congested.

It thus seems that at the outside there is only a certain *probability* that lead paralysis is of peripheral origin.

On the other hand, the reaction of degeneration and atrophy pursue exactly the same course as in spinal paralysis, and especially in the so called spinal paralysis of children; and it is highly probable that the same alterations occur in this disease, in the nerves and muscles, as in lead paralysis, although the point has not yet been systematically investigated.

The spinal origin of the paralysis thus appears to be a *possibility;* and I am at present inclined, on various grounds, to regard it as more probable than the peripheral origin, partly on account of its obvious analogy with the spinal paralysis of children, with regard to distributive atrophy of the muscles and electrical relations, partly on account of the regular symmetrical affection of the same nerve regions, partly on account of the merely partial paralysis of definite fibres of the same nerve trunk (the supinator longus remaining free), and lastly on account of the sensibility remaining unaffected.

I do not attach much importance to the objection that the careful examinations hitherto made of the spinal cord, as by Gombault and Westphal (lead-poisoning), have only yielded negative results. It is conceivable that in saturnine intoxication there are certain trophic disturbances (motor-trophic) of the central apparatus, which are not observable under the microscope, but which are nevertheless capable of modifying the nutrition of the peripheral nerves and muscles in various ways. Furthermore, we have to deal with foci of disease of small extent, which easily escape observation. It must not, therefore, be forgotten, I think, that a spinal origin of lead paralysis is possible. Future researches should be directed especially to the condition of the peripheral nerves, which should be followed, on the one hand, into the muscles, and, on the other hand, through their roots to the spinal cord, which should itself be very carefully examined, since the focus of the disease is probably of very limited extent. Similar researches should also be conducted in cases of spinal paralysis of children and in progressive muscular atrophy.

The *symptoms* of paralysis of the musculo-spiral nerve are extremely characteristic, and are recognizable by the practised physician at the first glance. The musculo-spiral nerve, as is well known, supplies the triceps muscle, and a small part of the brachialis anticus, and all the extensor muscles of the forearm. In accordance with this, we find that when this nerve is completely paralyzed, the hand is kept in a state of flexion; it hangs flaccid, and cannot be raised or extended; the fingers are flexed and cover the thumb, which is flexed and adducted. The patient is unable to extend the fingers, and when he attempts to do so the interossei and lumbricales alone act; and these, as is well known, only extend the two terminal phalanges, whilst they flex the basal phalanx. Neither the thumb nor the index finger can be abducted or extended; the patient is unable to supinate the forearm, at least when the arm is extended (in order to exclude the action of the biceps) (paralysis of the supinator brevis), neither can it be bent and half supinated by the supinator longus. The paralysis of this muscle is easily recognized by the circumstance that if the arm be placed in a half flexed and half prostrated position, and the patient be then directed to make a powerful effort to flex the arm, the hard projection of the supinator longus does not make its appearance, but its muscular belly remains flaccid and soft. If the triceps be concomitantly affected, the patient is unable to extend the arm with any amount of force, nor can he extend it vertically if the upper arm be raised. If the hand be laid flat upon a table, no lateral movements can be made with it, nor can it be raised from the surface of the table; whilst in this position, the lateral movements of the fingers, which are performed by the interossei, remain unimpaired; at the same time the mobility of the flexors appears to be interfered with, because the wrist can no longer be fixed, during their action, by the extensors, in consequence of which their points of insertion approximate to one another, and their contractions are feeble; but if the wrist be forcibly fixed in an extended position, it may be easily seen that the flexors are not in any way paralyzed.

The disturbances of the functions of the hand produced by paralysis of the musculo-spiral nerve, are more considerable than those of any other paralysis of the arm; the patient is almost entirely deprived of the use of the hand; on account of the thumb, and on account of the impaired functions of the flexors, he can neither hold nor grasp anything; he is unable to supinate the hand, and is entirely incapable of all fine work, and indeed of almost all coarser kinds of labor. The paralysis seriously interferes with almost all the numerous employments of daily life.

There is generally more or less intense anæsthesia in the region supplied by the musculo-spiral; if the cause of paralysis lie high, the anæsthesia affects the region of the superior and inferior posterior cutaneous nerves, and hence the posterior part of the upper arm and the extensor region of the forearm; if at a lower point, only the region of distribution of the same nerve in the hand (dorsal surfaces of the first three fingers as far as to the second phalanx, and the corresponding part of the back of the hand, and ball of the thumb) is affected. The slightness of the disturbance of sensation which accompanies complete motor paralysis is often very striking; it may indeed be entirely absent (Lannelongue, Savory, and Bernhardt). Such cases can only be explained by the fact that there are anastomoses between the large nerve trunks of the forearm, the existence of which was first conjectured from different observations made in surgery, and afterwards demonstrated to be a fact by the experiments of Arloing and Tripier upon dogs; to this anastomosis is due the at least partial preservation of sensation after section of a given nerve, in the region supplied by that nerve. There can be no doubt, however, that these communications are not equally numerous and extensive in all cases. The patients frequently only complain of a subjective sensation of numbness and furry feeling in the parts of the skin affected, without any objective diminution of sensibility being observable.

More or less complete atrophy of the extensor muscles, dependent upon the amount of lesion in the nerves, may not unfrequently be also observed.

In striking contrast to this, there appears in many instances a painless swelling of the extensor tendons over the wrist-joint. This is circumscribed, of the size of a hazelnut, elongated and movable. It was first observed by Gubler in lead paralysis, and has since then been frequently noticed. Gubler also saw this swelling in hemiplegic patients, and Nicaise and I found it in cases arising from injury. The symptom has been described by the name of tenosynitis hyperplastica, and undoubtedly owes its origin to the mechanical irritation which the tendons running over the wrist experience when that joint is strongly flexed. The view that the paralysis of the nerve also predisposes to this trophic disturbance, is rendered more probable by the circumstance that articular swellings are not unfrequently observed at the same time in the carpal and digital joints.

The electrical excitability varies, in the manner already described, according to the cause of the paralysis, and may here, as in other cases, render it possible for an estimate to be formed of the degree of trophic disturbance in the muscles and nerves, as well as afford some assistance in diagnosticating the seat of the paralysis. In the slighter forms of paralysis, arising from pressure, as from the use of crutches and in the so-called rheumatic paralyses, the electrical excitability is usually quite normal, and it may often, in such cases, be satisfactorily employed to determine the position of the cause of paralysis in the nerve, since below the point of lesion the excitability is normal, whilst no reaction can be obtained when the current is applied above it; and by this means the point of obstruction to the conduction may often be very exactly circumscribed. In one case of pressure-paralysis I found the previously-mentioned intermediate form of reaction of degeneration. Well-marked reaction of degeneration (with the characteristic alterations of faradic, galvanic, and mechanical excitability) occurs, in correspondence with the stage of the paralysis, in all severe traumatic musculo-spiral paralyses, and in lead paralysis, though here usually only in some of the muscles. In all instances the muscles present a high degree of atrophy. Lastly, in hemiplegic paralysis the electrical excitability is normal, or but slightly increased.

The *diagnosis* of musculo-spiral paralysis is uncommonly easy, and even paretic conditions of individual muscles supplied by this nerve are, as a rule, readily recognizable. The diagnosis of the nature and seat of the cause of paralysis is often a matter of greater difficulty; yet even here it is usually only a question of pressure, injury, or lead poisoning, since the other forms, as the cerebral, neuritic, and hysteric paralyses, can usually be easily recognized by the concomitant symptoms and the results of the anamnesis.

Ordinary *paralysis from compression* (and the so-called rheumatic paralysis) is characterized by the following symptoms: paralysis of *all* the muscles on the extensor side of the forearm, including the supinators, non-implication of the triceps, disturbance of sensibility *only* in the hand, the persistence of normal electrical excitability. *Crutch paralysis* is characterized by implication of the triceps, by the electrical excitability being normal, and by clear evidence of the cause. *In severe traumatic paralysis*, different muscles will be implicated in different cases, in accordance with the position of the wound. In many instances particular muscles escape remarkably—thus Bernhardt saw, in a case of traumatic paralysis from dislocation of the shoulder, the supinators remain unaffected ; and I myself met with a similar case in which the extensor digitorum was much more severely affected than the radialis and supinator longus. Other diagnostic symptoms of severe traumatic paralysis are the reaction of degeneration and atrophy of the muscles.

Lead paralysis.—We must dwell on the characters of this affection at somewhat greater length. It usually commences in the extensor digitorum communis, a few fasciculi of which are first affected, and then the whole muscle. In varying succession, the radial extensors, the extensor ulnaris, the extensors of the thumb, and the abductor longus pollicis are then affected. The *supinator brevis usually long remains unaffected, and the supinator longus escapes almost without exception, being only affected in very rare cases*, and then at a late stage and to a very moderate degree. This freedom of the supinators, though not absolutely a certain criterion, affords an extremely valuable means of diagnosis. Both arms are usually affected shortly after one another. The reaction of degeneration usually occurs early and very markedly in a part of the paralyzed muscles, coincidently with progressive atrophy. A few of the less severely affected muscles may, however, preserve their electrical excitability. In these latter, the motility, according to Duchenne, is rapidly recovered under appropriate treatment, whilst those affected with the reaction of degeneration always require a very long period for recovery. That other muscular regions, as the interossei, the muscles of the thenar eminence, the deltoid, the dorsal muscles, the muscles of the lower extremities, etc., may be affected by lead paralysis, can here be only indicated. In many cases the veins of the forearm, especially those of the extensor side, are remarkably swollen and varicose (Hitzig). The atrophy of the muscles, the absence of any disturbance of sensibility, the occurrence of tendinous swellings about the wrist, the presence of other symptoms of lead poisoning, especially the blue line on the gums, antecedent colic, etc., will all serve to establish the diagnosis.

The *course and prognosis* of musculo-spiral paralyses depend essentially upon the cause of the affection. The slight paralyses from compression usually occurring suddenly, as well as those of rheumatic origin, are often of remarkable duration; they seldom disappear in one or two weeks, but often last from four to six weeks, and not unfrequently for months; they may even, as observed by Brenner, persist for years. The motility then gradually returns, and the progress of the patient in regaining the power of raising the wrist and extending the fingers may often be followed from day to day. The prognosis is almost absolutely favorable, though even here unfavorable cases are occasionally seen. Paralysis from the use of crutches is still more favorable; recovery, if the cause be removed, and appropriate treatment adopted, usually taking place in from one to two weeks. The case of Busch, above cited, demonstrates that even very long persisting pressure on the nerve does not prevent the rapid restoration of its functions. Severe traumatic paralyses in the region of the musculo-spiral have the same tedious and protracted course that they elsewhere exhibit. Several months, and frequently from half a year to a year, elapse before recovery is complete; improvement may even continue after several years. But in most cases recovery occurs if the lesions of the nerve have not been too severe and irreparable. On these depend the prognosis.

Lead paralysis has also in all instances an exceedingly slow course, months usually elapsing before the more severely affected muscles commence to respond to voluntary impulses. Electrical investigation often supplies valuable information for the establishment of the prognosis. Even here, however, very severe cases recover in the course of one or two years, providing the cause of the poisoning be removed. It is rare that paralysis and atrophy progress continuously until death takes place. Like hysterical paralyses in general, hysterical paralyses of the musculo-spiral nerve vary much in their course, and have the same prognosis. Musculo-spiral paralysis, which is a very common symptom of cerebral hemiplegia, is characterized amongst the coincident paralyses of other nerve regions by its peculiar obstinacy. Extension of the fingers, of the wrist and thumb, are the last movements which such patients recover. Speaking generally, the prognosis of these paralyses resembles that of cerebral paralyses.

d. *Paralysis in the Region of Distribution of the Median Nerve.*

The median nerve is, upon the whole, rarely affected independently with paralysis. Rheumatic cases are very rare, but traumatic paralyses, such as may be occasioned by unskilful venesection, cuts, pressure, contusions, bad crutches, fractures of the humerus, gunshot wounds, etc., are far more frequent, whether affecting the nerve in the upper arm or its terminal branch above the wrist. Paralyses from neuritis and from neuromata also occur, and are sometimes observed after acute diseases; lastly, paralysis of the median is a very common symptom of central paralyses, whilst particular muscles supplied by the median, especially those of the thenar eminence, are often paralyzed as a consequence of progressive muscular atrophy.

The muscles implicated in this form of paralysis are the flexor carpi radialis, the pronators, the flexor digitorum sublimis and the external portion of the flexor digitorum profundus, the flexor pollicis longus, and, lastly, the small muscles of the ball of the thumb, with the exception of the adductor brevis. Flexion of the second phalanx is impossible in every finger; that of the third phalanx of the index and middle fingers is also impossible, and flexion and opposition of the thumb are quite impracticable. On the other hand, flexion of the first phalanx, with extension of the second and third by the agency of the interossei, can be very well performed in the four fingers; not unfrequently, owing to the excessive action of the interossei, a kind of hyperextension of the two last phalanges, especially of that of the forefinger, is effected. The patient is unable to perform opposition, flexion, or any of the more delicate movements of the thumb, which is permanently extended and adducted, and kept closely applied to the forefinger, as in the hand of the ape.

Flexion of the wrist is only practicable when the hand is strongly adducted by means of the flexor ulnaris, the action of the radial flexor being deficient. The patient is almost entirely incapable of pronating the hand, as this can only be performed in a very incomplete manner by the supinator longus. The three last fingers can still be partially bent, because the flexor profundus digitorum is in part supplied by the ulnar nerve. These various effects give to the hand and fingers, and especially to the thumb, a position so peculiar that isolated paralysis of the median can be immediately recognized. The high degree of atrophy of the paralyzed muscles in the forearm and ball of the thumb, which is not unfrequently present, further assists in diagnosticating this affection.

If disturbances of sensibility be present, they are exhibited in the lateral part of the palm of the hand, on the palmar side of the thumb, index and middle fingers, and in the ungual phalanx upon the dorsal side also. Disturbances of sensibility may be completely absent, even when the median is divided above the wrist (Richet[1]), and this is explicable by the already mentioned anastomoses which the median forms with the other nerves of the forearm (Arloing and Tripier). I have, moreover, seen the sensibility of the affected parts of the skin completely lost, with coincident paralysis of the median and ulnar nerves above the wrist, although the radial was completely intact; and in one case of section of the median above the wrist, there was complete anæsthesia in the affected fingers, although the ulnar and radial were completely uninjured. In the three first fingers, trophic disturbances of the skin and nails not unfrequently make their appearance in severe paralyses of the median, such as glossy fingers, ulceration, pemphigus vesicles, abnormal growth of hair, etc.

In regard to the electrical relations, the diagnosis, prognosis, and course of median paralysis, the same general observations hold as in cases of radial paralysis.

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[1] Duchenne, however, found in this case that the electro-muscular sensibility was lost. Electrization localizée, 3. édition, p. 350.

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e. *Paralysis in the Region of Distribution of the Ulnar Nerve.*

Although this nerve appears, from its superficial position in the upper arm, and above the wrist, to be much exposed to injury, it is nevertheless not very frequently affected with paralysis. The etiology of the paralyses of this nerve is nearly the same as that of median paralyses ; traumatic causes, such as pressure, contusion, gun shot and punctured wounds, fractures of the humerus, dislocations of the shoulder, pressure of crutches, and sleeping upon the arm placed beneath the head, being the most frequent. I have seen paralysis of the ulnar nerve occasioned by a small neuroma above the elbow, which had originated in consequence of frequent mechanical injury of the nerve at this point. Duchenne saw this disease frequently in workmen who in their occupation rest the elbow firmly on a hard support. Progressive muscular atrophy affects by preference the small muscles of the hand which are supplied by the ulnar.

Paralysis of the ulnar nerve affects the flexor carpi ulnaris, the greater part of the flexor profundus digitorum, all the muscles of the hypothenar eminence, together with the interossei, a part of the lumbricales, and the adductor pollicis brevis. In correspondence with this, the following movements are impaired : Ulnar flexion and adduction of the hand are limited; complete flexion of the three last fingers is rendered difficult or impossible ; the ability to move the little finger is almost entirely abolished ; separation and lateral compression of the fingers against one another, as well as flexion of the first and extension of the second and third phalanges of all the fingers, are rendered impossible, owing to the paralysis of the interossei. If the interossei and lumbricales are alone paralyzed, the traction of the extensor communis and of the two flexors (extension of the first and flexion of the two last phalanges) produces that remarkable claw-like position of the hand, which is so well-known and characteristic a symptom of paralysis of the ulnar above the wrist, and of certain cases of progressive muscular atrophy; this is at the same time more strongly marked, in cases of ulnar paralysis, in the two last fingers than in the second and third, because their lumbricales are supplied by the median nerve; lastly, the patient is unable to adduct the thumb and apply it firmly to the metacarpus of the index finger.

As a consequence of these various disturbances of motility, the functions of the hand are materially interfered with, the finer and more delicate movements which are required for writing, drawing, and playing upon the piano, being more or less impaired; at the same time, the use of the hand is not entirely abolished, as motility is preserved in the muscles supplied by the radial and median nerves. If, however, as frequently occurs, the muscles of the thenar eminence or part of the extensors be coincidently affected, the use of the hand is almost entirely abolished.

Disturbances of sensibility, if present, affect the well-known region of distribution of the ulnar nerve, namely, part of the palm of the hand, the palmar surface of the fourth and fifth fingers, part of the back of the hand, and the dorsal surface of the three last fingers; the same observations apply here in regard to its presence or absence as in the case of the musculo-spiral and median paralyses.

Trophic disturbances (especially the characteristic atrophy of the interossei, and the depression of the intermetacarpal spaces), and the electrical relations of the paralyzed nerves and muscles, are similar to those that occur in the paralyses of the two other nerves of the forearm.

The diagnosis of ulnar paralysis does not present the smallest difficulty, as the failure of the power of adduction in the joint of the hand and the paralysis of the interossei are particularly characteristic symptoms of the affection. The seat and causes of the paralysis must be determined on general principles. Secondary contractures, if any be present, are easily recognized. In regard to the prognosis and course, the observations made in regard to musculo-spiral paralysis may be referred to.

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Treatment of the Paralyses of the Upper Extremity.

The removal of the causes of the disease should first be attempted, and, if effected, will prove of great advantage. Surgical means are not unfrequently requisite before any other treatment can be adopted; and amongst such measures may be mentioned the reduction of dislocations and fractures, the removal of abnormally deposited callus (Busch), the excision of compressing cicatrices, and ultimately the surgical reunion of divided nerves, the treatment of all kinds of wounds, the removal of tumors, which exert pressure, and of neuromata,—these all constitute measures of the highest importance in particular cases. Little can be done for the treatment of rheumatic paralyses, though counter-irritants, diaphoretics, and the iodide of potassium may be tried. Antiphlogistics and the application of galvanic currents are useful when neuritis is certainly present. In hysterical and central paralyses it is essential that the primary disease should be treated. In lead paralysis it is of the greatest importance that the patient should avoid further absorption of lead by giving up or interrupting for a time his business or occupation, or that he should adopt proper preventive measures. The poison should then be eliminated as soon as possible by the usual means, baths, sulphur baths, iodide of potassium, and improvement of the general health. In chronic cases of traumatic paralysis, and in paralyses resulting from articular rheumatism, or from chronic neuritis, improvement may not unfrequently be obtained by the employment of malt- and mud-baths, and by taking the baths of Wildbad, Teplitz, Wiesbaden, brine baths, etc.

Amongst the means that may be directly applied to the relief of the paralysis, electricity is alone deserving of confidence, since it not only exerts a powerful direct anti-paralytic action, but also fulfils the causal indication. In regard to the application of the faradic or galvanic currents, the strength of the current, the place, duration, and frequency of the application, the general rules already laid down (page 459) are completely applicable. In severe traumatic paralysis, long-continued or repeated applications of the current, which should by preference be the galvanic current, are requisite. In slight paralyses from compression (as of the musculo-spiral nerve from the use of crutches) faradisation is upon the whole tolerably successful, though even here it appears to me that the galvanic current frequently exerts a greater and more rapid influence. The galvanic current is of most service in lead paralysis, although faradisation appears to have been followed by excellent results in Duchenne's practice. In addition to local galvanization, which should be applied to all the affected muscles, and if possible to the musculo-spiral nerve throughout its whole extent, galvanization of the cervical portion of the spinal cord may be recommended, and it remains for further researches to determine whether galvanization of the sympathetic is not also useful in the treatment of this form of paralysis. In all instances the plan of treatment adopted must be both persistently and systematically carried out.

Little benefit can be obtained from the use of alcoholic and irritating liniments or from external measures generally. On the other hand, in many cases active or passive gymnastic exercises, pursued with energy and perseverance, may materially hasten the progress of recovery.

After this description of the several forms of paralysis, affecting the upper extremity, it still remains to say a few words in regard to their more frequent combinations. These are indeed very manifold, and it is easily conceivable that in the multifarious combinations that take place between the nerves, forming the brachial plexus, great diversity should be presented, especially in the paralyses of the plexuses, in the grouping of the particular nerves and muscles affected, according as one or other fasciculus of the brachial pexus is chiefly affected by the cause of paralysis.[1]

In this respect the paralyses that occur after dislocations of the shoulder, exhibit great variety. It is evident that, in subcoracoid luxations, the immediately subjacent nerve-trunks of the brachial plexus are especially liable to injury, and, according to circumstances, the whole of the nerves may be compressed or lacerated, or one or more may remain wholly intact, or be but slightly damaged. Hence the variable picture presented by these paralyses. In one case the arm will be completely paralyzed, the circumflex, musculo-cutaneous, and the three nerve-trunks of the forearm, being equally affected by paralysis. In another case only the circumflex and musculo-spiral nerves are paralyzed, whilst in other cases again the biceps and brachialis anticus remain intact, or motion may be preserved in the flexors of the forearm, whilst all the other muscles are paralyzed. In almost all these instances the paralysis originates in severe injury. The reaction of degeneration is exhibited in the affected nerves and muscles, and recovery is exceedingly slow. In those nerves, however, that are more slightly affected, excitability is preserved, and in these the mobility usually soon returns, though it is, no doubt, not uncommon for particular severely injured nerves to remain permanently paralyzed.

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[1] Seeligmüller has observed symptoms of paralysis of the cervical sympathetic, namely contraction of the fissure of the lids and of the pupil of the same side, in two cases of paralysis of the brachial plexus ; and it is quite likely that these symptoms are characteristic of many cases.

In paralyses from fracture of the humerus the result is dependent upon whether one or several of the nerve trunks of the forearm have been injured, or are subsequently implicated in the development of the callus. The musculo-spiral is most frequently paralyzed, then the ulnar, and more rarely the median nerve. In these cases the paralysis is generally of the severe traumatic kind, and recovery is exceedingly slow.

Dislocations of the elbow-joint and fractures in its vicinity and of the forearm constitute common causes of paralyses of the nerves of the forearm, and it is evident that the ulnar and the median are particularly liable to be affected. After these injuries paralysis frequently arises in consequence of improperly or too firmly applied bandages, as I have several times observed, both in nerve trunks above the wrist and above the elbow. These are always very obstinate and protracted, and not unfrequently incurable paralyses.

I have, on several occasions, observed a peculiar *combination of paralyzed muscles* in spontaneously arising (rheumatic) as well as in traumatic paralyses in the arm: namely, coincident paralysis of the deltoid, biceps, and brachialis anticus, as well as of the supinator longus. Here and there other muscles were also affected, so that in two cases the supinator brevis was also implicated; whilst in another all the muscles supplied by the median in the forearm were affected, etc. The above-named muscles, however, were always chiefly, if not exclusively, affected. This indicates that the seat of the lesion is at a point where the fibres forming the circumflex, musculo-cutaneous, and a part of the musculo-spiral nerves lie in close proximity to each other. In fact, it is possible, by very careful faradic excitation of the several branches of the brachial plexus, to succeed in discovering, in many individuals, a spot (which corresponds to about the point of emergence of the sixth cervical nerve between the scaleni), from which the deltoid, the biceps, brachialis anticus, and the supinator, may be thrown into common and very energetic contraction. (It is at the same time often difficult to avoid the immediately adjacent origin of the musculo-spiral nerve; if this be separately excited, the muscles supplied by this nerve contract, with the exception of the supinator longus.) I am consequently of opinion that the form of paralysis here mentioned has its seat in the root of the brachial plexus at this point, and recommend that further attention should be paid to it, and, that when an opportunity occurs, it should be anatomically investigated. I have observed it, in two instances, as a consequence of neuritis—once in consequence of a fall upon the arm and shoulder, and once in consequence of a cancer in the glands of the neck and in the vertebral column. The electrical excitability was, in the last case, normal, in the others there was more or less well-marked reaction of degeneration; all terminated in recovery. In two cases the patients complained of a sensation of numbness in the region of distribution of the median in the hand.

It is very remarkable that about the same muscles are found to be paralyzed in that form of paralysis which Duchenne has described[1] in newly-born children, as "paralysie obstétricale infantile du membre supérieur,'' and which we should call "delivery paralysis'' (Entbindungslähmung). I have observed two cases of this affection, and can corroborate Duchenne's description in all essential points. The subjects are always children who have been brought into the world by obstetric operations, by turning and subsequent extraction, or by traction applied to the shoulders in cases of difficult delivery of the body. In one case, the paralysis appears to be induced by the use of the finger as a hook, in the axilla ; in another case it may be attributed to the difficult disengagement of the arm in the process of delivery; and, lastly and especially, as it appears to me, the paralysis owes its origin to the energetic application of the so-called Prague grip, in which the fingers are applied like a fork over the back of the neck, and endanger the brachial plexus by energetic traction and compression. Such children are born with paralysis, though this is not always immediately discovered, as part of the upper extremity still remains movable. The paralysis, however, always presents very characteristic features; the arm hangs immovable by the side of the body, it is rotated inwards, and is persistently extended. The child is unable either to flex the forearm or to raise the arm, yet the movements of the hand and fingers are preserved. On careful examination, which presents very considerable difficulties in such little patients, it may be shown that the deltoid, the biceps and brachialis anticus, and the infraspinatus, with the teres minor and perhaps the supinators are paralyzed. Sensibility of the skin does not appear to be materially impaired. The electrical excitability is usually diminished (reaction of degeneration ?). Contractures often make their appearance at an early period in the non-paralyzed muscles, as, for example, in the pectoralis major, in one of my cases. The prognosis of this very characteristic paralysis, which cannot easily be mistaken for any other form, and which exists without any complication arising from luxation or fracture, is not very favorable. Neglected cases, in many instances, do not recover at all ; and on this account Duchenne recommends that faradic treatment, from which he has obtained excellent results, should be applied at an early period ; appropriate galvanic treatment might, perhaps, be equally useful. There can be no doubt that this form of paralysis arises from mechanical compression applied during delivery to a part of the brachial plexus.

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[1] Electrisation local., 3 ed. p. 357.

Paralysis of the upper extremity may, however, arise during delivery in another way. Duchenne mentions (l. c.) two cases in which paralysis of the brachial plexus arose from the pressure of the awkwardly-applied forceps, and one case where paralysis of two nerves of the forearm was occasioned by fracture of the humerus occurring during delivery, and he lastly draws attention to the frequent occurrence of subacromial dislocations of the humerus, during delivery, as a cause of more or less extensive paralysis of the brachial plexus. Careful attention paid to these occurrences and a careful investigation into this class of cases will, without doubt, narrow the somewhat obscure domain of "congenital" paralyses.[1]

8. *Paralysis of the Muscles supplied by the Lumbar and Sacral Nerves.*

A. Eulenburg, l. c. pp. 595—602.—*Brenner*. Unters. u. Beobacht. etc. II. p. 203.—*Bianchi*, des paralysies traumatiques des membres infér. chez les nouvelles accouchées. Thèse, Paris, 1867.—*Duchenne*, Electr. loc. 3ᵉ éd. pp. 983 et seq.; pp. 1016 et seq.—*Nothnagel*, die nervös. Nachkrankheiten des Abdom. typhus. Arch. f. klin. Med. IX. 1872. Consult also the Manuals of Electrotherapeutics and the Treatises on Diseases of the Spinal Cord and Vertebral Column.

Paralyses in the region of the lower extremity, the muscles of which are supplied by the branches of the lumbar and sacral plexus, are of very frequent occurrence, and are due to very various causes. Central paralyses, however, are undoubtedly those which possess most practical interest. Almost all spinal paralyses commence in the lower extremity, and hence peripheral paralyses have been much less frequently reported, and have received less attention. We shall here endeavor to give a short sketch of the symptoms of the several forms of paralysis and of their more frequent causes, and must refer for further details to the account of diseases of the spinal cord.

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[1] See Seeligmüller. Berl. klin. Woch. 1874. Nos. 40 and 41.

1. *Paralysis in the Region of Distribution of the Crural Nerve.*

Isolated paralysis of the crural nerve is not of very common occurrence. It results from injuries of the vertebral column and pelvis, from tumors and extravasations of blood in the cauda equina, not unfrequently in consequence of inflammation of the knee-joint, after which the whole region of the extensors of the leg often become atrophic and paralyzed. It also occurs in consequence of inflammation of the psoas, and psoas abscesses, preceded by antecedent symptoms of irritation ; frequently also in consequence of fractures of the thigh and dislocations of the hip-joint ; not unfrequently, also, from cuts, stabs, and gunshot wounds of the lower abdominal and crural regions. Neuritis again is often a cause of crural paralysis, as are also pelvic and crural tumors. Lastly, it is a very frequent symptom of all forms of spinal paralysis, and more rarely of cerebral paralyses and of progressive muscular atrophy.

Symptoms.—Paralysis of the crural nerve affects the ilio-psoas muscle, the quadriceps extensor femoris, the sartorius, and the pectineus. The symptoms are consequently very uncomplicated ; the patient is unable to flex the leg at the hip-joint or to raise the body from the recumbent posture. He is unable to extend the lower leg, or, when sitting, to move the vertically hanging lower leg forwards. Standing and sitting upright are rendered insecure ; walking, jumping, and running are either rendered difficult or impossible, because in all these actions the flexors of the thigh and the extensors of the leg are necessarily used. When the paralysis of the crural affects both sides, the patient finds it almost impossible to walk forwards.

Disturbances of sensibility are frequently observed, and, when the crural nerve is itself affected, may extend over the lower two-thirds of the thigh, the region of the knee, and the inner side of the lower leg, as far as the internal border of the foot. If the inguinal region, the hypogastric region, the scrotum, and the external surface of the thigh are rendered anæsthetic, this indicates that the seat of the cause of paralysis is situated above the giving off of the cutaneous branches affected (ilio-hypogastric, ilio-inguinalis, lumbo-inguinalis, spermaticus externus, and cutaneus femoris lateralis) from the plexus lumbalis. Sensations of furriness, numbness, and frequently also an objectively demonstrable chilliness of the thigh are commonly present.

Atrophy of the muscles of the thigh is very frequently present, sometimes only ascertainable by very careful, comparative measurement, but generally very well marked, so that the contour of this part of the limb is materially modified. The atrophy as well as the paralysis are often only partial, and limited to particular muscles and parts of muscles, which then appear flaccid, relaxed, and diminished in size, whilst the others, especially when efforts are made to innervate them, or when faradic irritation is applied, project strongly, and become hard with sharply defined outlines.

Electrical examination indicates changes which vary with the kind and degree of the paralysis, and of the consecutive trophic disturbances which have been already frequently described, and which we need not here further discuss.

2. *Paralysis in the Region of Distribution of the Obturator Nerve.*

This is of still rarer occurrence than crural paralysis, but is frequently associated with the latter, and is then referable to the same causes. In addition to the causes mentioned, may be added compression of the nerves from strangulated obturator hernia, and from the pressure of the descending head of the child or of obstetric instruments in difficult deliveries.

This paralysis affects the adductors of the thigh, the pectineus, the gracilis, and the obturator externus. The patient is consequently incapable of adducting the thigh, of pressing the knees together, or of crossing one leg over the other. The flexion of the thigh inwards is rendered difficult, and so too is the rotation of the thigh outwards; the affected leg soon tires in walking; some disturbances of sensibility are perceptible extending down the inner side of the thigh as low as the knee. It is difficult to recognize partial paralyses of particular muscles belonging to the group of adductors. The electrical relations have not been sufficiently investigated in these paralyses.

3. *Paralysis of the Muscles supplied by the Glutæal Nerves.*

This form of paralysis is also rare, occurring almost exclusively as a symptom of extensive paralyses, affecting the whole region of the sacral plexus, as, for example, in those due to tumors and lesions of the cauda equina, to fractures of the sacrum and pelvis, to spinal diseases, etc. The characters of progressive atrophy, and of pseudo-hypertrophy of the muscles are not unfrequently completed by atrophy and paralysis of the glutæal muscles.

The paralysis effects the glutæal muscles, the tensor fasciæ, the obturator internus, and the pyriformis, and is thus capable of expressing itself in various motor disturbances of the manifold and complicated functions of these muscles, which may, however, be in part compensated by the action of other muscles, and are consequently analyzed with difficulty. The rotation of the leg, both inwards and outwards, is interfered with; flexion of the thigh is somewhat limited and its abduction is rendered remarkably difficult, or altogether impossible. Some uncertainty is felt both in standing and walking, because the glutæal muscles, and especially the glutæus maximus, fixes and balances the trunk upon the thighs; the patient finds it particularly difficult to ascend stairs. The straightening of the body, after it has been bent forward, is rendered difficult. Disturbances of sensibility are only present when other nerves are coincidently affected. Atrophy of the muscles is of common occurrence, and, when the paralysis is unilateral, is very evident. The glutæal region loses its firmness and roundness, the muscles appear relaxed and feeble, and allow the subjacent parts of the skeleton to be readily perceived. The electrical excitability can only be tested by intramuscular excitation, but then exhibits alterations corresponding to the cause of the paralysis and the secondary disturbances.

4. *Paralysis of the Muscles supplied by the Sciatic Nerve.*

The wide distribution of this nerve, its exposed and superficial position in various parts of its course, and its manifold relations to the pelvic organs, render the frequency, on the one hand, and the importance, on the other, of the paralyses affecting it, intelligible. The paralysis may affect the nerve as a whole, or either of its two branches, the peroneal and tibial nerves, or only particular muscles. The paralysis is very frequently bilateral.

Etiology.—The most frequent causes of paralysis of the sciatic nerve are mechanical lesions of the trunk, or of the branches, whether they result from gradually increasing pressure or sudden contusion, or from laceration or section, and it is difficult to give an exhaustive catalogue of all the modes in which such an injury may be inflicted. Paralyses of this nerve and its branches have been observed to follow injuries caused by gunshot wounds, cuts, and punctures at the most different points; after surgical operations, such as extirpation of tumors, tenotomy, etc.; after fractures of the spine, after falls or blows upon the glutæal region; after extravasations of blood in the cavity of the sacrum; as a result of tumors in the cauda equina; in consequence of mechanical compression of the nerves at any part of their course; after difficult deliveries, in which especially the nerves of the sacral plexus, which occupy the posterior part of the pelvis, are damaged by the pressure of the head of the child; after dislocations of the femur; in newly-born children after delivery by the feet; as a result of the compression of cicatrices, as I have seen in one case of deep-seated cicatrix, following a bed-sore; as a result of tumors of the pelvic cavity (cancers and fibroid tumors in the uterus) or of the nerve itself, etc.

Paralysis of the sciatic is much less frequent as a result of "rheumatic" causes, such as thorough wetting and chilling of the feet and of the buttocks. Those arising from well-marked neuritis are of common occurrence, to which also, probably, belong the not unfrequent paralysis and atrophy remaining after severe sciatica. We are still insufficiently acquainted with the anatomical changes which often persist after acute diseases, and which lie at the root of the paralyses which develop spontaneously in the region of the sacral plexus, without demonstrable cause, and without well-marked symptoms. This is true also in regard to the hysterical paralyses of the lower extremity that are of such frequent occurrence. This nerve region commonly participates in the various spinal and in very many cerebral paralyses. The region of distribution of the sciatic nerve is relatively rarely affected in progressive muscular atrophy, whilst it almost constantly participates in the pseudo-hypertrophy of the muscles.

Symptoms.—We shall now consider the symptoms of paralysis of the several branches of the sciatic nerve, out of which the picture of the total paralysis of the nerve can be easily formed.

If the *peroneal nerve* be alone affected, the anterior muscles of the lower leg are paralyzed. The foot hangs flaccidly down, it can neither be flexed nor abducted, and can only be incompletely adducted; walking is seriously interfered with, in consequence of the depending point of the foot, which trips upon every little elevation of the ground. The patient is compelled to give the necessary elevation to the foot in moving forwards by flexion of the hip-joint, and plants it vacillatingly and insecurely with the point and outer border of the foot first, producing a very characteristic, and, for this paralysis, pathognomonic gait. In its further course, this peculiarity is rendered still more striking by secondary contracture of the muscles of the calf.

The part played by the several muscles in producing these symptoms is as follows: paralysis of the tibialis anticus considerably limits the dorsal flexion and adduction of the foot; the inner border and the point of the foot can no longer be completely raised, though these movements may be in part vicariously executed by the extensor digitorum communis and the extensor longus pollicis. Paralysis of the extensor digitorum communis likewise diminishes the dorsal flexion of the foot, and the abduction of the foot in the flexed position, whilst it, at the same time, renders extension of the basal phalanges of all the toes impossible. Paralysis of the extensor pollicis longus diminishes dorsal flexion, and abolishes the power of extending the great toe. Paralysis of the peroneal muscles acts differently; if the peroneus longus be paralyzed, abduction of the foot in the extended position is impossible ; the arch of the foot, which is essentially preserved by the action of this muscle, becomes flattened, but the inner border of the foot no longer touches the ground, because the head of the first metatarsal bone is no longer drawn downwards (Duchenne); a peculiar kind of flat foot is thus produced, which has been described with great care by Duchenne. If, lastly, the peroneus brevis be paralyzed, pure abduction of the foot is rendered impossible ; this can only be then accomplished either with coincident dorsal flexion, by means of the extensor digitorum communis, or with coincident plantar flexion, through the agency of the peroneus longus. Paralysis of the little extensor digitorum communis brevis somewhat impairs the extension of the basal phalanges of the four last toes. All thes evarious forms of paralysis may occur in an isolated manner, but they may also be combined in the most various ways, and, as the defective movements may, also, to some extent, be compensated for by other muscles, very complicated conditions arise, the nature of which can only be ascertained by the exercise of great care and special investigation—the difficulties of the case being often materially increased by secondary contractures and anomalous positions of the foot.

If the *tibial nerve* be paralyzed, the whole of the muscles at the back of the lower leg are rendered inactive ; this is immediately indicated by the inability of the patient to affect plantar flexion (extension) of the feet, as well as flexion and lateral movement of the toes. Paralysis of the *musculus triceps suræ* (gastrocnemius and soleus) prevents the foot from being extended, and renders it impossible for the patient to stand upon the toes, etc., whilst, in consequence of secondary contracture of the muscles of the front of the lower leg, a hook-like position of the foot is gradually produced. Paralysis of the *tibialis posticus* diminishes the power of adducting the foot and of raising its inner border. Paralysis of the *flexor communis digitorum* renders flexion of the two distal phalanges of the toes impossible. Paralysis of the *flexor hallucis longus* takes away the power of flexing the great toe. Paralysis of the *adductor and abductor hallucis* abolishes the power of moving the great toe laterally, whilst paralysis of the *interossei*, just as in the hand, renders flexion of the first, and extension of the two distal phalanges of the toes, as well as separation of the toes, impossible. A peculiar claw-like position, resembling that of the hand, is thus produced. The first phalanx is abnormally extended; the second and third are strongly flexed; the toes are strongly compressed together, and no longer touch the ground with their bulbous extremities. The functional disturbance produced by these conditions is relatively small; some pain and inconvenience are, however, experienced after long standing or walking, because the heads of the metatarsal bones have now to sustain the weight of the body.

Independently of the disturbances of particular movements or of all the movements of the foot, which interfere to a greater or lesser extent with walking and standing, these paralyses of the muscles of the lower leg occasion very remarkable anomalies in the position of the foot, secondary alterations of the joints, and malformations, which may gradually lead to persistent and most annoying conditions. It is especially paralyses of particular muscles (as, for example, of the peroneus longus of the tibialis, of the triceps), which occasion various forms of paralytic flat foot, pointed foot, hook foot, and club foot. From the above description it is evident how the several paralyses, especially with the aid of coincident contractures of the antagonists, and under the influence of the weight of the body, dispose to various forms of curvature of the foot. It is obvious that these are particularly likely to occur in growing individuals, in whom the bones and ligaments are not yet consolidated, and hence an explanation is afforded of the special frequency of such paralytic curvatures as a result of the spinal paralysis of children. Duchenne has made a special study of all these disturbances, and we are indebted to him for having greatly elucidated their pathogenesis. Want of space prevents us from entering into the details, and the whole subject is one rather of surgical and orthopædic interest. If the trunk of the sciatic nerve be affected, the branches distributed to the flexors of the lower leg, the semi-tendinosus, semi-membranosus, and biceps femoris are also paralyzed, and the patients are not able to flex the lower leg upon the thigh, to approximate the heel to the glutæal region, or to offer any resistance when an attempt is made to extend the leg. Rotation of the thigh is also interfered with because the branch for the quadratus femoris, and sometimes also that for the obturator internus, is given off above from the trunk of the nerve. But even when the paralysis of the sciatic is complete, walking is still, to some extent, possible, because the leg is simply used as a stilt, and with the aid of the muscles of the thigh is moved forwards, the knee-joint being kept fixed in the extended position. The whole foot hangs placid, the foreleg is strongly raised in locomotion forward, the external border of the foot is first placed upon the ground, and it is then made a support by extension of the knee. This is, of course, a very imperfect mode of walking, but it may be learned by the patient after a little practice, even when the paralysis of the sciatic is bilateral, as I have frequently seen.

Paralyses of the sciatic are usually accompanied *by disturbances of the sensibility*, the extent of which depends on the cause of the paralysis. When the paralysis is limited to the perineal region, the anæsthesia is limited to the anterior and external side of the lower leg, the dorsum of the foot and the greater part of the toes; if the tibialis is affected, the posterior surface of the lower leg, the sole of the foot and plantar surface of the toes, are the seats of the anæsthesia. According as the cause of paralysis is situated at higher planes in the trunk of the sciatic, the region of the knee, the back part of the thighs, and ultimately the buttock and perinæum, are successively affected by anæsthesia. If the cause of the paralysis is seated within the cavity of the sacrum, or, still higher, in the cauda equina, the anæsthesia affects the whole sacral region, the scrotum and penis (or the labia, as the case may be), the urethra, bladder, and rectum. These symptoms may prove of great importance for the local diagnosis.

Disturbances of the circulation, usually in the form of stasis, cyanosis, bluish-red marbled coloring, and coldness of the skin, are often perceptible in the paralyzed leg. Increase of temperature has been occasionally and transitorily observed in the earlier periods of traumatic paralyses.

Trophic disturbances are also not unfrequent in cases of severe peripheral paralyses of the sciatic.[1] Amongst these may be mentioned a high degree of atrophy, ulceration of the skin, eruption of herpes and pemphigus, serious bed-sores on the sacrum, ankles and heels. A very disagreeable concomitant symptom is, lastly, *paralysis of the rectum and bladder*, which is especially liable to occur in cases where the cause of the paralysis is situated within or above the sacrum in the cauda equina. It is sufficiently well known that these symptoms, with all their evil results, are very common in the various spinal paralyses of the lower limb.

A description of the *electrical relations* in these paralyses would only lead to a repetition of what has been already stated in the general part. The most diverse anomalies occur, and they suggest the same conclusions as in paralyses generally.

The *diagnosis* of paralysis in the region of distribution of the sciatic nerve and its branches presents no notable difficulties. The recognition of the paralyses of particular muscles in the lower leg, their diagnosis from contractures, the determination of the degree of trophic disturbance they have undergone, etc., alone require, in many instances, careful and cautious investigation. A very exact comparison should be made between the legs of the affected and of the healthy side; the shape of the foot in standing, walking, and in ordinary positions, should be noted, and the state of the several muscles should be examined by means of electricity, and each movement of which the extremity is capable should be separately performed, in order that every relation may be rendered clear. Conclusions should be drawn in regard to the nature of the cause of the paralysis, partly from the anamnesis and partly from objective investigation conducted upon general principles. Evidence of the plane at which the cause of the paralysis acts on the motor nerves implicated may be derived from the local extent of the paralytic symptoms in the sensory and motor nerves, the concomitant symptoms that may happen to be present, the alteration of the electrical excitability, and the relations of the reflex actions, etc.

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[1] In one case of well marked unilateral paresis of the tibialis and peroneus, I observed a distinct hypertrophy of the muscles of the calf.

It is of great importance to determine whether we have to deal with a peripheral or with a spinal paralysis. I have met with various cases in which it was impossible to determine this point with certainty; in most cases, however, a correct diagnosis may be made with a little care. Various circumstances help us in forming an opinion; thus, incomplete paralysis of particular muscles, their gradual attack, the degree of sensory disturbance not being of equal extent, weakness of the bladder occurring at an early period, the existence of muscular tension, tremors, and the like, the presence of other special symptoms (such as a feeling of a girdle round the waist, swaggering gait when the eyes are closed, tenderness of the vertebræ), preserved or increased electrical excitability, and the occurrence of paretic symptoms on both sides, are all circumstances that point to the spinal origin of the affection. If reflex actions are preserved in the paralyzed muscles, it is certain that the seat of paralysis is centric, whilst, upon the other hand, the absence of reflex actions is by no means a proof that the affection is not spinal. The existence of the reaction of degeneration is in favor of the peripheral seat, providing corresponding sensory disturbances are present at the same time. Reaction of degeneration without any disturbance of sensibility is in favor of the spinal origin of the paralysis (spinal paralysis of children, etc.). Compare, in addition, what has been said above (page 444) on the differential diagnosis of centric and peripheral paralyses.

The *prognosis* of these paralyses is, in general, doubtful; it, of course, essentially depends upon the cause, but secondarily upon the consecutive trophic disturbances, the electrical relations, etc. It must also be remarked that in consequence of the great length of the nerves under discussion, the processes of regeneration in them may require a very long time before the conducting paths to the muscles are re-established. Thus, in traumatic and similar paralyses, we must be prepared for a very long course of the affection, and often for its incurability. In other respects the prognosis must depend on general principles.

Treatment of Paralyses of the Lower Extremity.

We may here refer to what has been said in regard to the upper extremity. In addition to the causal treatment, in which the treatment of diseases of the spinal cord plays an important part, it is electricity, of course, to which we should resort in most instances, especially since it appears to be able to fulfil many of the causal indications. There is nothing especial to say in regard to the choice of the methods of its application, though in most instances, since it is frequently desired to influence the nerves within the spinal column and pelvis, the galvanic current is to be preferred, because the faradic has no noticeable influence upon such deeply situated tissues.

In the next place, the application of a rational and steadily practised system of gymnastics may be recommended. Baths, whether thermal, saline, or in the form of mud-baths, must be employed only according to special indications. Little benefit can be anticipated from embrocatious of any kind; but even such weak means as these will not be omitted, in tedious cases, to obtain the confidence of the patient.

Orthopædic surgery plays a very important part in these paralyses, partly by effecting the removal of deformities or preventing their development, and partly by supplying the function of the paralyzed muscles by means of appropriate apparatuses, and by restoring to the patients, if only artificially, the use of their lower extremities in standing or walking. A great deal can be done in this direction, and for details the reader is referred to treatises on orthopædic surgery.

ANATOMICAL DISEASES OF THE PERIPHERAL NERVES.

1. *Hyperæmia of the Nerves.—Congestion.*

Weir Mitchell, Injuries of nerves. Phila. 1872, p. 56. *A. Waller*, On the sensory, motory, etc. symptoms resulting from the refrigeration of the ulnar nerve. Proceed. Roy. Soc. of Lond. XI. p. 436. XII. p. 89. 1862.

Little is known in regard to active hyperæmia of peripheral nerves; treatises on pathological anatomy are silent on this point, and we know little or nothing of the clinical symptoms presented by this condition, which is probably often present. Still it appears to be deserving of some attention in the future, since, perhaps, many so-called functional neuroses are referable to it.

Weir Mitchell is the only author who has given a short description of hyperæmia; he employed artificial freezing and thawing of the tissues, as an experimental means of investigating it, and has not only frozen the partially exposed nerves of animals by ether spray, but also the ulnar nerves of living men. From these observations it appears that the symptoms occurring and remaining for some time after the thawing are to be ascribed, not so much to the freezing and thawing themselves, as to the subsequent hyperæmia.

Anatomical examination made in experiments on animals showed that, after thawing, the nerves presented more or less extensive rosy or dark-red injection, which often had a linear or striated marking, whilst the nerve appeared somewhat swollen. It was only after violent action of cold that small punctiform extravasations of blood were discoverable between the nerve fibres.

Symptoms.—After the disappearance of the symptoms, which are due to the freezing (pain, anæsthesia, paralysis, increase of temperature and augmented perspiration in the region of distribution of the nerve), and which have been especially studied by Haller, there occurs, in the first place, in the thawed portion of the nerve a very *painful sensibility*, which spreads backwards to the plexus of origin, and in some instances may even produce reflex vertigo and feeling of faintness. With this is associated *hyperæsthesia* in the whole region of the affected nerves, a *feeling of numbness*, *creeping*, and *formication, a certain motor debility*, and a *sensation of fulness in the affected part*, together with slight swelling; no longer any notable elevation of temperature.

Where this group of symptoms occurs in man in the region of distribution of a definite nerve, and when, at the same time, perhaps a spot, sensitive to pressure, can be demonstrated in the nerve, we are justified in admitting the existence of active hyperæmia of the nerve.

In regard to *treatment*, Mitchell recommends the energetic application of ice to the whole length of the nerve, an elevated position, and absolute rest of the part; in severe cases a large number of leeches should be applied, which may, however, produce a good deal of pain, on account of the hyperæmia present; the subcutaneous injection of morphia and atropine, and, at a subsequent period, the effect of digitalis and quinine may be tried.

2. *Inflammation of the Nerves.—Neuritis.*

Rokitansky, pathol. Anat. 3. Aufl. II. p. 498. 1856.—*Förster*, Handb. d. path. Anat. 2. Aufl. II. p. 498. 1863.—*O. Weber*, Pitha-Billroth. Handb. d. Chir. II. 2. p. 214. 1865.—*Hasse*, l. c. p. 739.—*M. Rosenthal*, Nervenkrankheiten, 1870, p. 457.—*Nasse*, Diss. de neuritide Hal. 1801.—*Swan*, Diss. on the treatm. of morbid local affect. of nerves, Lond. 1820.—*Descot*, Affect. local. des nerfs. Par. 1822.—*Martinet*, Revue méd. Juin, 1824.—*Abercrombie*, Pathological and Practical Researches on Diseases of the Brain and Spinal Cord. Edinburgh, 1829.—*J. B. Friedreich*, über die Localkrankheiten d. Nerven. Schmidt's Jahrb. V. p. 89. 1835. (Complete list of earlier authorities.)—*Dubreuilh*, Rech. expérim. sur l'inflamm. des nerfs. Clin. de Montpell. 1845, Nos. 5. u. 7.—*Bérard*, Note sur les accid. qui suiv. la piqu. des nerfs. Journ. des connaiss. méd.-chir. 1846, Mars.—*Beau*, Arch. génér. 1847, and Union méd. Juill. 1849.—*Piorry*, de l'hemito-névrite et de son traitement. Union méd. 1851, Nos. 79 u. 80.—

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Remak, über Neuritis. Oesterr. Ztschr. für prakt. Heilk. 1860, Nos. 45—48. —*Duménil*, Contrib. pour serv. à l'histoire . . . . . de le névrite. Gaz. hébdomad. 1866, Nos. 4—6.—*W. Erb*, Pathol. u. path. Anat. periph. Paral. (Neuritis facial.) Arch. f. klin. Med. IV. V. 1868.—*E. Tiesler*, Ueber Neuritis. Diss. Königsb. 1869.—*Virchow*, Neurit. interstit. prolifer. Virch. Arch. Band 53, 1871.—*J. Althaus*, Neuritis des Plex. brach. Arch. f. klin. Med. X. 1872.—*Weir Mitchell*, Injuries of nerves, etc. Philad. 1872. p. 66.—*O. Wyss*, z. Kenntniss des Herpes Zoster. Arch. d. Heilk. XII. 1871.—*Bernhardt*, z. Pathol. d. Radialisparalys. Arch. f. Psych. u. Nerv. IV. 1874.

The frequency and significance of the presence of neuritis is still a subject of dispute, chiefly because the indifferent anatomical methods of research hitherto employed do not supply satisfactory evidence of the nature of the affection, and still more because the symptoms do not always demonstrate with sufficient exactness the existence of inflammation of the nerves. If some (as, for example, Remak and Benedict) go too far in making acute or chronic neuritis answerable for the most diverse nervous symptoms and groups of symptoms, and even for numerous trophic disturbances in other tissues, the opinion of others, who maintain that neuritis is of rare occurrence, can not be regarded as any longer tenable. Neuritis, even when of spontaneous origin, is probably of more frequent occurrence than is generally admitted, and if the views recently suggested by several weighty authorities, in regard to the mode of extension of neuritis and its disposition to affect the central organs of the nervous system, should be only partially corroborated, the results of neuritis would still possess an importance in nerve pathology that could not be overlooked. The affection, therefore, deserves careful consideration.

Etiology.—The most frequent and best known causes of neuritis are *wounds* of various kinds—cuts, stabs, punctured wounds (in former times not unfrequently injury of the median in venesection), contusion, rupture, laceration, the penetration of foreign bodies into the nerves. Slight mechanical injuries may also cause neuritis, such as a blow on a nerve trunk, strong compression of a nerve, severe concussions of the nerves from long travelling in an ill-constructed wagon (Duménil), sudden and violent muscular movements, violent efforts to raise heavy weights, etc. Traumatic neuritis has been observed to arise from all these as well as from many other forms of accident.

No doubt can be entertained that in many instances neuritis arises from "*catching cold;*" exposure to a draught of air, for example, wet feet, sudden chilling of the body, standing or walking in cold water, etc., have all been observed to constitute undoubted causes of this disease.

Inflammations of neighboring organs, extending to nerves traversing or adjoining such organs, constitute a very common cause of neuritis. Beau has demonstrated this in regard to the intercostal nerves in cases of pleurisy, pleuro-pneumonia, and tuberculosis of the lungs ; and his observations have been repeatedly corroborated in numerous cases of acute and chronic inflammatory and suppurative processes. Thus both the acute and the chronic form of articular rheumatism, especially of the shoulder, not unfrequently lead to propagated neuritis. The adjoining nerves are often inflamed in cases of caries of the temporal and sphenoid bones in caries of the vertebræ, and of the bones of the pelvis ; progressive neuritis has also been observed to be caused by inflamed tendinous sheaths. Malignant neoplastic formations, such as carcinoma and sarcoma, penetrate among the nerve trunks, and set up neuritis.

After *acute diseases*, such as typhoid fever, the acute exanthemata, diphtheria, etc., neuritic processes are not unfrequently developed, as has been recently demonstrated by Bernhardt, anatomically, in the musculo-spiral nerve in a case of exanthematic typhus. *Many chronic diseases* likewise lead to inflammatory alterations in the peripheral nerves, as is seen in syphilis. Alterations which may be regarded as inflammatory are also exhibited by the nerves in lepra anæsthetica (perineuritis chronica leprosa).[1]

The question still remains open whether neuritis can be induced by *separation of the nerves from the central nervous system.* It is well known that the histological changes that take place in the peripheral segment of a nerve, after division, present great similarity to inflammatory alterations, and are often spoken of as being really neuritic. We have already attended to the question (see p. 422) whether these alterations are to be explained as a direct propagation of the affection from the seat of the lesion, or as the result of the so-called trophic influences, and must here again repeat that we consider the separation from the central nervous system to be the essential cause of these alterations; hence the question whether they are really to be regarded as inflammatory must still be looked upon as an open one. Further investigation pursued upon a right basis will, no doubt, elucidate the existence and kind of influences exerted by the centre upon trophic and circulatory disturbances in peripheral regions.

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[1] Virchow, Geschwülste. Band II. p. 521.

Lastly, no one can wonder that it is impossible in many cases to discover the real cause of neuritis, and hence that we must speak of its origin as spontaneous. It appears, moreover, as if many persons have a special predisposition to neuritis, and especially as if the disposition for the disease to spread upwards or downwards along the nerves is present in very different degrees in different individuals. Special attention must be paid to this point in the future.

Pathological Anatomy.—In acute neuritis the nerve is swollen and deeply reddened, exhibits increased succulency, has a cloudy aspect, and loses its peculiar refractive powers. The nerve fasciculi are surrounded by a serous or even fibrinous coagulable exudation, forming a curdy yellowish red mass, in which small punctiform extravasations are not unfrequently visible. Microscopical examination discloses marked congestion, a moderate amount of emigration of white corpuscles into the neurilemma, which is swollen with serous exudation, and incipient disintegration of the medulla of the nerve fibres. At a subsequent period well-marked suppuration and softening occur; the nerve becomes soft, easily lacerated, and sometimes converted into a chocolate-colored pulp. The nerve fibres are completely broken down, and even the axis cylinders are completely lost. The nerves occasionally undergo actual sloughing. In the earlier stages the inflammatory process may be limited, and recovery may take place; the hyperæmia disappears, the emigrated elements are carried away, or undergo fatty degeneration and absorption, the serous exudation vanishes, the degenerated nerve fibres undergo regeneration, and the nerve is gradually restored to its normal state. It is more common, however, if death do not supervene, for the neuritis to pass from the acute into the chronic form.

In this *chronic neuritis*, which frequently commences as such, the disposition to the formation of pus is less marked, and there is a greater tendency to neoplastic formation of connective tissue, and to induration and sclerosis. The nerve appears to be thickened, sometimes uniformly, for considerable distances, sometimes in a nobbed or spindle-shaped manner, and not unfrequently exhibits several such swellings at varying distances from one another—*neuritis nodosa;* the nerve becomes tougher, of cloudy aspect, and of a whitish gray color, passing through leaden gray into violet; it becomes more or less intimately adherent to the adjoining parts; the thickening and adhesion frequently affect only the external sheath of the nerve, so that, though confined and compressed, it is still movable in its sheath; more frequently, however, the whole is converted into a homogeneous mass of connective tissue, in which the neurilemma and perineurium as such are lost, and which separate the nerve fasciculi to a greater or less extent from each other. The hyperæmia is moderate, seldom high in degree, and still less frequently so intense as to lead to small extravasations of blood. Microscopical examination shows congestion of the vessels and interstitial accumulation of cells, remarkable increase of interstitial connective tissue, granular cells, fatty degeneration of the nerve medulla, and ultimately atrophy and utter destruction of the nerve fibres, including the axis cylinders. On transverse section these changes are found to have taken place unequally, well-preserved nerve fibres being visible amongst others that present all stages of regressive metamorphosis. In many cases the growth of the connective tissue so predominates histologically that we may admit, with Virchow, a *neuritis interstitialis prolifera.* No remarkable changes occur in the segment of nerve situated centrally to the seat of inflammation; the normal characters are, as a rule, presented at a distance of only a few lines above this point; on the other hand, in the peripheral portion, as soon as well-marked degeneration of the nerve fibres (and probably strong compression of them) has taken place in the focus of the inflammation itself, the well-known degenerative changes which we have already described in extenso, occur in the nerve, and, if the nerve affected be one of mixed endowments, in the muscles also. Amongst these may be mentioned interstitial cell and tissue proliferation, degeneration and disappearance of nerve fibres, atrophy of and nuclear growth in the muscular fibres, etc. Subsequently the already mentioned trophic disturbances in the skin, bones, nails, etc., may occur, just as in paralyses arising from wounds, severe compression, and rheumatism.

Recovery may ultimately take place at the very seat of the disease itself; this, however, in all instances occurs but slowly. The retrogressive changes of the hypertrophied connective tissue and the regeneration of the destroyed nerves require time. The process, for the most part, advances to connective-tissue induration, to sclerosis and complete atrophy and degeneration of the nerve fibres. The nerve then appears to be converted into a tough gray and often pigmented connective-tissue cord, which is ultimately fused with the adjoining parts. The division between the sheath and the nerve is no longer distinguishable; the whole appears to be converted into a homogeneous mass of tough wavy connective tissue, in which it is impossible to discover any trace of nerve fibres, except here and there an isolated one, which has passed into a state of degeneration or regeneration. A hyperplasia of the connective tissue, and even of the still preserved nervous tissue (neuritis hyperplastica, Ferréol-Reuillet), which may occasionally lead to false or true neuroma, is not unfrequently associated with this termination of the affection.

It still remains to mention that an extension of the disease from its original seat not unfrequently takes place, both downward and upward (*neuritis descendens* and *ascendens*). The more important extension in the centripetal direction occurs either uniformly and continuously, or in a sudden metastatic manner. In some cases, again, whilst certain parts of the nerve retain their normal character, others present circumscribed hyperæmia, swelling, and hypertrophy of connective tissue (Froriep, Rokitansky, Tiesler, and others), and in this way the spinal cord may ultimately become affected (Tiesler, Feinberg).

Symptoms.—The symptoms vary according to whether the neuritis is acute or chronic. *Acute Neuritis:* In this form, shortly after exposure to one or other of the causes of the disease (traumatism, sloughing, development of cancer), well-marked feeling of chilliness, or an actual rigor is experienced, which marks the commencement of sharp fever, accompanied by headache and sleeplessness. Pain, quickly augmented in intensity, and proceeding from the affected nerve, and spreading over its region of distribution, and frequently over the whole member, indicates the cause of the affection. This pain is generally intense, persistent, deep-seated, tearing, boring, and burning, with frequent remissions, and usually with nocturnal exacerbations. It is augmented by every movement of the limb and by everything that excites the circulation. In excitable patients it may, according to W. Mitchell, produce a high degree of agitation of the whole system, slight delirium, and a kind of hysterical condition. It radiates into other nerves of the same plexus, and in severe cases into more remote nerves, as, for example, from the brachial nerves to the fifth nerve.

In some cases a *red line* is observed in the skin over the course of the nerve. The nerve is always extremely sensitive to pressure or pinching. It can often also be felt to be distinctly swollen. The skin over the nerve, and in the whole area of its distribution, exhibits a *marked degree of hyperæsthesia*, whilst, at the same time, there are subjective sensations of *numbness* and formication; anæsthesia appears at a later period, when the rapidly formed exudation begins to compress the nerve fibres. Coincidently with this there are symptoms of *muscular debility*, and as in the early period the great pain in the nerve inhibits movement, so distinct paretic symptoms now set in, which may increase to complete paralysis. The temperature of the skin has been observed to be augmented in the region of distribution of the affected nerve. The occurrence of œdema and of profuse sweating in the same region has also been noted.

This condition is not persistent, if the affection be not complicated with tetanus, and we cannot enter into further details on this point. All the symptoms may subside with tolerable rapidity, and complete recovery set in; but it more often passes gradually into the subacute and chronic form.

Chronic Neuritis.—So many and such a variety of symptoms have been ascribed to this affection, that it is difficult to discriminate those which are essential and are proper to the neuritis, from those which are unessential, secondary, and more of an accidental nature.

The symptoms of chronic neuritis make their appearance either after the previous occurrence of acute symptoms, or quite spontaneously, often very insidiously appearing in a slight manner for weeks or months, and then disappearing, and either gradually or suddenly rising to great intensity.

Pain is usually the most constant, as it is the earliest symptom; it is described as being more or less continuous, and exhibits, in different instances, considerable differences in character and intensity, sometimes being only dull and tensive (as Bean describes it in the upper intercostal spaces of phthisical patients), at others of considerable violence, interrupted by exacerbations of a lancinating, tearing character, and always radiating towards the periphery. Not unfrequently it assumes the character of a well-marked neuralgic attack, the exacerbations almost always occurring at night, and preventing sleep; it is increased by all kinds of exertion and movement, and by every excitation of the activity of the heart.

Coincidently with the pain, or even, in some insidious cases, before the occurrence of the pain, paræsthesia occurs, the patient complaining of numbness, creeping, formication, and of unpleasant pricking sensations, if the skin is touched or struck.

More or less well-marked *motor symptoms of irritation* are observed, according to the violence of the attack, such as a disagreeable tension of the muscles, tremors, sudden contractions, and more rarely violent tonic cramps and persistent contractures; these symptoms are, in some instances, conjoined with weakness of the muscles, and in all nerve regions *symptoms of paralysis* occur. It may reasonably be admitted that the above-mentioned symptoms of irritation accompany the stage of hyperæmia, commencing exudation, and emigration of white corpuscles, whilst the consecutive symptoms of depression are caused by the augmenting proliferation of the interstitial tissue, by compression and progressive degeneration of the nerve fibres.

Anæsthesia of various grades occurs in this stage, from slight blunting to complete loss of sensibility for the various kinds of sensation, to which may be superadded motor pareses that not unfrequently rise to complete paralysis. It is remarkable for how long a time, in many cases of neuritis, the motor nerves remain unaffected after the conductivity of the sensory nerves has long been impaired, though occasionally the opposite condition is observed—early and predominating paralysis of the motor nerves. (See cases reported by Bernhardt.)

*Symptoms of radiation and various reflex phenomen a*are very common, especially in the stage of irritation, and when the disease pursues a subacute course. Pain extends upwards into the various branches of the same plexus (even when the neuritis does not creep any farther upwards), and more unfrequently into remote nerve regions. Reflex phenomena occur first in those muscles that react normally to reflex irritation, and subsequently in more remote nerve regions. Reflex cramp is often very violent, so that, for example, the nails of the fingers bury themselves in the skin of the hand from cramp of the flexors (Stromeyer). Meyer observed a case resembling writers' cramp, occurring as a result of neuritis of the musculo-spiral nerve. In rare cases there are general convulsions, hysterical convulsive attacks, and even well-marked tetanus.

Swelling of the affected nerve, if it happen to be accessible to palpation, may be felt, and this may either be diffused over a considerable portion of the length of the nerve, or be fusiform or moniliform. The nerve is *always very sensitive to pressure*, and not temporarily only, as at the painful points of Valleix in neuralgia. Pressure made upon the swelling not unfrequently occasions excentric sensation (pain, formication).

The *electric relations* of nerves affected with neuritis have not been investigated with sufficient care. In the stage of irritation and in slight cases the excitability, both for galvanic and faradic currents, is exalted. I have observed this very distinctly in a case of neuritis of the median nerve. If the paralysis be well marked (and in such cases only is the electrical examination usually made), the electrical excitation may remain normal, as in many slight paralyses proceeding from compression; but if the disease have advanced to degeneration of the nerves, the reaction of degeneration is always present, as Bernhardt observed, and as I have likewise seen in several cases. In one case of neuritis of the brachial plexus I found the above-mentioned middle or intermediate form of degeneration. (See p. 437.)

Many other symptoms make their appearance that seem to be more or less closely connected with the neuritis, though the evidence of their relation to it has not as yet been satisfactorily obtained. Amongst these are the phenomena proceeding from compression and subsequent degeneration of the nerves, and from the consequent degeneration of the muscles; also a high degree of *atrophy of the muscles*, with the reaction of degeneration, etc. In regard to the question whether these symptoms are the consequence of the neuritis, *per se*, or are only the results of the absolute suppression of conduction in the nerves, as has been already mentioned, cannot at present be stated with certainty.

Nor is our information more satisfactory in regard to the *trophic disturbances* in the skin and nails, the swelling and stiffness of the joints, that have not unfrequently been observed in neuritis. Remak, in particular, has drawn attention to the dependence of many articular affections upon neuritic conditions, and Benedict corroborates his statements. Remak has also referred certain forms of progressive muscular atrophy, which commences in the muscles of the thumb, to neuritis of the brachial plexus, and has already endeavored to explain the subsequent affection of the opposite arm by an extension of the process across the spinal cord. In the face of known facts, no doubt can exist in regard to the dependence of herpes zoster upon neuritic processes; but the question still remains open, whether the zoster originates from an irritation of trophic nerve centres, as Bärensprung, Wyss and others suppose, or whether, as Friedreich believes, it is due to the ultimate extension to the tissues of the skin of a descending neuritis. In the case reported by Virchow, epilepsy occurred, apparently due to the irritation of the inflamed nerve, and disappeared after excision of the affected part of the nerve. This had previously also been frequently observed. The history of traumatic trismus and tetanus is also, as is well known, frequently referable to neuritis.

Duration, course, and terminations.—Acute neuritis lasts for a few days or weeks. It either proceeds at once and steadily to improvement and recovery, or, as is more frequently the case, it passes into the chronic form.

Chronic neuritis is a disease of quite indeterminate but always protracted duration. Many weeks or months pass by before recovery takes place from even the slightest forms. (See, for example, Waller's case of neuritis, caused by freezing of the ulnar nerve.) The traumatic forms are the most uncomplicated, especially when produced by a simple cut or puncture. As soon as the reunion of the nerves has taken place, the disturbances of sensibility first disappear, the motility then recurs, and ultimately the atrophy and any secondary disturbances of nutrition that may have been produced gradually vanish, and thus a complete *restitutio ad integrum* may be effected. The period required for this fortunate result, no doubt, varies considerably, the greater or less perfection of the recovery being essentially dependent on the accidental nature of the disease. Idiopathic forms of neuritis, or those dependent on rheumatic causes, or occurring after acute diseases, are less favorable. They may last for months or for years, and, in some instances, never entirely disappear, since exacerbations follow every slight provocation, such as movement, exertion, exposure to cold, etc. Persistent neuralgia and anæsthesia, debility, and even complete motor paralysis are not unfrequently sequelæ of such a neuritis. Still, even in such doubtful cases, improvement and recovery are sometimes observed to occur at a late period. If the neuritis be occasioned by disease of neighboring organs (inflammation, suppuration, new formation, etc.), its course is determined essentially by the relation of the primary affection. If this can be rapidly and completely set aside, the neuritis may be expected to subside gradually; whilst, if it be incurable, it leads to thorough softening and destruction of the nerve, or it passes into a state of complete sclerosis; recovery is then impossible.

It is obvious that the symptoms and the course of neuritis will vary according to whether the nerves affected are purely sensory, purely motor, or of mixed formation. In each case the symptoms may be easily deduced from the general description that has already been given. It is also easy, from the account formerly given of the neuralgiæ, cramps, and paralyses of the several nerves of the body, to anticipate the symptoms of neuritis affecting each nerve trunk.

A point of special importance, in reference to the course of neuritis, and to the whole form and complexion of many particular cases of disease, is one noticed by various observers, namely, the *disposition of the neuritis to propagate* itself along the nerve, chiefly in a centripetal direction, and to extend to nerves lying at a higher plane, and even to the spinal cord. Earlier observers, Rokitansky, Froriep, and others, had already admitted such a centripetal mode of propagation of neuritis. Remak describes neuritis as having a special tendency to progress in an ascending and also in a descending direction, and refers to this cause a number of diseases. Weir Mitchell also attributes to neuritis a great disposition to extend centripetally. Leyden[1] is inclined to refer the myelitis occurring after affections of the urinary and other pelvic viscera, to a lumbo-sacral ascending neuritis, extending to the spinal cord; and Remak also regards the paraplegia occurring in such cases as being due to lumbo-sacral neuritis. Duménil has observed wide-spread chronic myelitis result from primary ascending chronic neuritis. Ferréol-Reuillet[2] also describes a remarkable case, in which a neuritis hyperplastica, resulting from fracture of the humerus, propagated itself to and even beyond the spinal cord. Lastly, Friedreich[3] has paid special attention to ascending neuritis, and regards it as a middle term between primary myositis and the (secondary) affection of the spinal cord in progressive muscular atrophy, and, in addition, refers numerous other morbid processes either to ascending or descending neuritis.

There are other facts, however—and they have been already mentioned (p. 400)—which appear to prove that inflammatory states may develop in the spinal cord from peripheral neuritic foci without the intervention of an ascending neuritis. We refer to the observations of Leyden, to the experiment of Tiesler, and to the results of the researches of Feinberg.[1] Our information is still imperfect in regard to the mechanism of these inflammatory conditions extending from the periphery to the central organs. Nevertheless, it would appear from the above briefly mentioned facts, which are worthy of particular attention, that the neuritis may be unexpectedly complicated with serious centric disturbances, and that it, perhaps, affords a means of explaining the connection between many hitherto obscure morbid processes.

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[1] Volkmann's Sammlung klin. Vorträge, No. 2, 1870.

[2] Virchow-Hirsch, Jahresbericht, for 1869. Band II. p. 349.

[3] Ueber progressive Muskelatrophie, u. s. w. Berlin, 1873.

The *diagnosis* of neuritis does not, in general, present any remarkable difficulties ; where pain and paræsthesia occur in the area of distribution of a certain nerve, where there are symptoms of motor and sensory irritation, with subsequent paralysis, and where, finally, painful swelling of the nerve can be made out, the diagnosis cannot long remain doubtful. Acute neuritis, in particular, is not easily mistaken, especially when, as is usual, it is connected with an injury inflicted upon a nerve, or in the neighborhood of a nerve. On the other hand, the recognition of chronic neuritis, especially of its insidious forms, is often very difficult. It may be very easily mistaken for neuralgia, and as long as it is possible to ascribe all neuralgiæ to neuritis, as long as it is impracticable to frame a sharp definition of neuralgia, the distinction between the two must always be more or less arbitrary. It is ordinarily admitted that the term neuritis is appropriately applied to those cases where the pain is more or less continuous, where the distinctly circumscribed painful points of Valleix are absent, and where indications of sensory and motor paresis are of early occurrence. I have, however, seen cases of well-marked chronic neuritis (in sciatica) where the pains presented a distinctly intermitting character.

Neuritis is readily distinguished from *muscular rheumatism* by the seat and extent of the pain, and by the pain being felt when certain definite movements are performed, and when pressure is made on certain muscles. Many difficulties, however, arise when the neuritis affects nerves supplying muscles (trapezius, serratus major, deltoid, etc.).

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[1] Berlin klin. Wochenschrift, 1871.

Thrombosis and *embolism* of the larger vessels of the extremities, which often present symptoms similar to those of neuritis (as, for example, in phlegmasia alba dolens), may be recognized by the coincident disturbances of the circulation, by the œdema, necrosis, etc.

The diagnosis of neuritis from *diseases of the central organs of the nervous system* (excentric neuralgiæ, anæsthesiæ, paralyses, etc.) is effected by a consideration of the symptoms already given in describing these conditions.

The *prognosis* of neuritis is always somewhat doubtful, partly on account of its long duration, partly from the secondary symptoms of paralysis and trophic disturbance, and partly on account of the possible transference of the morbid processes to the central nervous system (producing epilepsy, tetanus, myelitis, etc). The forecast of the acute traumatic form is relatively favorable, as is also that of the circumscribed subacute and chronic forms which result from injury to the nerves. Idiopathic forms, or those originating in rheumatic conditions, are frequently very obstinate and tedious; only the slighter cases are likely to terminate favorably, though I have seen recovery take place in serious cases, accompanied by complete paralysis, well-marked reaction of degeneration, and atrophy. In every case, the prognosis must depend on the causes and extent of the lesion, and especially upon the individual peculiarities that may be present. The possibility of the neuritis extending by continuity must always be kept in view.

Treatment.—In the first place, the treatment must be directed to the removal of the cause, and surgical proceedings now occupy a prominent position, as in the removal of foreign bodies, cleansing and careful coaptation of wounded surfaces, disinfection of wounds, reduction of inflammatory symptoms, reduction of luxations and fractures, evacuation of abscesses, removal of carious bone, etc. So, too, the treatment of articular rheumatism, inflammation of tendons, syphilis, and other morbid conditions, which have been referred to as causes of the affection, must not be neglected. If the affection appears to be dependent on recent rheumatic disease, benefit may be obtained from active diaphoresis, from derivation through the skin, and from the administration of iodide of potassium.

If *acute neuritis* has really been established, energetic antiphlogistic treatment must be adopted: suitable applications of ice, free local depletion, purgatives, favorable position of the parts, and absolute rest. Relief from pain is obtained by the subcutaneous injection of morphia and atropine. Large doses of quinine may also sometimes be serviceable.

In the *subacute and chronic forms*, the activity of the antiphlogistic treatment pursued must be proportionate to the violence of the symptoms. Cold may be applied in the form of elongated ice bottles covering the nerve throughout its whole length; or the wet bandages of Priessnitz may be used, with subsequent employment of cool hip-baths. Local abstraction of blood is seldom requisite; whilst, on the other hand, derivatives and counter-irritants are often very beneficial. The effects of the application of the faradic brush over as large a surface as possible may be tried, as well as painting with iodine, repeated blistering, though not immediately over the affected nerves. Derivation from the bowels and skin, by means of purgatives and diuretics, is frequently indicated.

The sovereign remedy for all the more chronic forms is the *galvanic current*. It has been warmly recommended by Remak, who has frequently applied it with brilliant success. Leyden, M. Mayer, Althaus, and others, have corroborated this favorable action, and I have myself obtained very satisfactory results with it. The best mode of employing it is to keep the anode steadily applied to the affected spot every day (or less frequently), for a few minutes; improvement usually sets in rapidly, and is considerable. Other modes of application may, however, be required to meet the special circumstances of the case.

If the symptoms are at all severe, absolute rest of the affected part is necessary, which should be obtained by appropriate position. In all cases, indeed, rest and preservation of the part from injury, are very essential points in the treatment. The patient should be forbidden to take exercise, or to perform any kind of work requiring exertion, especially if involving exposure to cold water. Neglect of this precaution is often the cause of tedious progress and relapses.

In very obstinate and chronic cases recourse may be had to energetic counter-irritants (moxa, actual cautery), to hot baths, such as those of Wilbad, Gastein, Teplitz, Wiesbaden, to mud baths, and strong saline baths, and to vigorous cold-water cures.

The treatment of the symptoms, as they arise, also requires careful consideration; and we may refer on these points to what has already been said in the sections devoted to neuralgia, anæsthesia, cramp, and paralysis.

In regard to the treatment of the secondary centric symptoms, such as epilepsy, tetanus, myelitis, the chapter devoted to these points must be consulted.

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3. *Atrophy of the Nerves*

Rokitansky, l. c. II. p. 493.—*Förster*, l. c. Bd. II. p. 641.—*Hasse*, l. c. 2. Aufl. p. 750.—*M. Rosenthal*, l. c. p. 453.—*Jaccoud*, Atrophie nerveuse progressive. Leç. de Clin. méd. Paris, 1867, p. 372.—*Vulpian*, Influence de l'abolit. des fonct. des nerfs sur la région de la moëlle, etc. Arch. d. Physiol. norm. et path. 1868, No. 3, p. 443. 1869, No. 6, pp. 678 u. 690.—*Dickinson*, on the changes in the nervous syst. which foll. the amputat. of limbs. Journ. of Anat. and Phys. No. III. Nov. 1868.—*Th. Leber*, Beitr. z. Kenntniss d. atroph. Veränderung des Sehnerven, etc. Arch. f. Ophthalm. XIV. 2. p. 164.—*A. Eulenburg*, über vasomotorische und trophische Neurosen. Berl. klin. Woch. 1873, No. 2.

From what has been already said, it is abundantly clear that atrophy of the peripheral nerves is by no means of rare occurrence; but it has always hitherto been seen as a secondary affection or sequela of other diseases, as of wounds, compression and inflammation of nerves. Idiopathic, primary atrophy of the peripheral nerves, does, however, sometimes though very rarely, occur. We know little with certainty of its clinical history. The practical importance of this disease is consequently at present very small.

Pathological anatomy affords little assistance in solving these difficulties, and at present no means exist of diagnosticating a primary atrophy from one of secondary origin, arising from inflammation, or compression, or from central disease; still many observers are inclined to admit, as cases of atrophy, those in which the following conditions may be noted: simple disappearance of the nerve fibrils without fatty degeneration of the medulla, moderate hypertrophy of connective tissue with deposition of corpuscula amylacea, and a gray, semi-transparent aspect of the nerve, which, besides, will be found to have diminished in size.

This form of atrophy, which not unfrequently occurs in the optic nerve (Leber), must be distinguished from senile atrophy, in which, although the nerves become attenuated, drier, poorer in their watery constituents, and more rigid, their histological characteristics remain unaltered. This is, indeed, only that form of atrophy which affects many other tissues in advanced age. A somewhat similar condition is met with in the atrophy from general macrescence and cachexia, in which the nerves, like the other tissues, though to a less extent, participate. In this case also, apart from the mere diminution in volume, no characteristic anatomical change can be demonstrated.

Secondary atrophy, of the existence of which no doubt is entertained, and which has already (p. 413) been fully described, presents very different characters. Here fatty degeneration, and ultimately absorption of the medullary sheath, breaking up and disappearance of the axis cylinder takes place, with persistence of pale, fine fibres, which are collapsed nerve sheaths; in the interstitial tissues there are abundant plastic infiltration, granule cells, and occasionally corpora amylacea; the atrophy is often masked by the hypertrophy of the connective tissue. On examination with the naked eye, the nerves appear as pale gray, translucent, slender, flattened bands; their sheath is flaccid, often thickened and coalesced with the surrounding tissues. Here, therefore, there is well-marked degenerative atrophy, which is almost always preceded by fatty metamorphosis; it appears doubtful whether, as Foerster and some others admit, a direct atrophy may take place without antecedent fatty metamorphosis.

One of the most essential and most important conditions for the production of secondary atrophy appears to be *the arrest of* *the trophic influence normally proceeding from the central nervous system.*

For even when exercising all due care in weighing the numerous facts that are in part opposed to each other, and are in part capable of opposite explanations, it may be regarded as in the highest degree probable, if not absolutely certain, that some kind of influence—though this cannot be exactly defined—proceeds from the central organs, which preserves intact the nutrition of the peripheral parts, especially of the nerves and muscles, and the abolition or alleviation of which disturbs the nutrition of these, and ultimately leads to degeneration and atrophy.

Unfortunately, we at present possess but little information in regard to these trophic influences, either as to their anatomical seat or relations or their mode of action. It may, perhaps, at most be surmised that these data are to be sought for in the gray substance of the spinal cord and medulla oblongata, and perhaps, also, in certain limited areas of the brain. It is probable that the parts which are to be regarded as trophic centres are ganglion cells situated in the anterior columns of the spinal cord.[1] The mechanism of these actions is still very obscure, and even their very existence has been contested, and, very recently, from a quarter to which all deference should be paid.[2]

It has been suggested that all the forms of trophic disturbance occurring in consequence of central and peripheral nerve lesions, are referable to simple inflammatory processes in the nerves, to ascending and descending neuritis.

We consider the view, that trophic influences proceed from the centres, as the more probable one, and believe that it is capable, without straining, of explaining all the facts.

On this view it is easily intelligible why nerve atrophy invariably supervenes when these trophic centres have suffered from disease, or when they have been from any cause separated from the peripheral nerves. Experience, in fact, shows that such atrophies occur:—

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[1] See A. Eulenburg, loc. cit.; Kussmaul, in Volkmann's Sammlung klin. Vorträge, No. 54; Charcot, klin. Vorträge; Deutsch. von Fetzer, 1874.

[2] Friedreich, Ueber progressive Muskelatrophie, u. s. w.

1. *In consequence of central diseases*, as in anencephalic monsters and in spina bifida (Rokitansky), in paralytic idiocy, in bulbar paralysis, in many forms of myelitis, in tabes dorsalis, in the spinal paralysis of children, and in the analogous affections of adults (also in progressive muscular atrophy, if this, as most authors admit, is to be regarded as a spinal disease ; perhaps, too, saturnine paralysis belongs to the same category). Whether the atrophy of the optic and other cerebral nerves, which is so common in tabes dorsalis, is to be regarded as a secondary atrophy dependent on disease of the spinal cord, or as an isolated localization of the same process as the gray degeneration in the spinal cord, is not as yet ascertained. Are the trophic centres for these nerves also situated in the spinal cord ?

2. *In consequence of peripheral lesions*, as those arising from section or division of the nerve in any way (interrupting its continuity), from energetic compression of it by tumors of all kinds (aneurisms, cancerous growths, osteomata, syphilomata, etc.), or from neuritic processes, in which last, however, a direct trophic disturbance at the inflamed part itself may be admitted.

Another but less important condition for the production of secondary atrophy is *the destruction or abolition of the functions of peripheral organs*. In consequence of this, there occurs in a limited number of cases a centripetally extending atrophy of the nerves, which at first consists only of a certain amount of wasting and attenuation, but subsequently presents the characters of degenerative atrophy and gray degeneration. That form which occurs in the optic nerves is best known. Here simple atrophy is met with at an early period after extirpation, destruction and phthisis of the globe of the eye, after atrophy of the retina, after tumors in the eye, and after glaucomatous conditions (Leber), whilst at a later period the optic nerves become gray and transparent, and present the histological characters of gray degeneration (Leber). These changes appear not to extend, as a rule, beyond the chiasma. In a similar manner, many years after amputation of an extremity has been performed, a centripetally progressive atrophy of the divided nerves has been observed, often extending even to the spinal cord, and appearing either as a simple attenuation, without histological change (Vulpian), or with all the signs of a neuritic atrophy, hypertrophy of connective tissue, etc. (Dickinson). This, however, is by no means constant (Friedreich). Lastly, Wundt has observed, after atrophy of one kidney of several years' duration, a high degree of atrophy of the renal nerves.

The conditions that lead to the production of primary atrophy are not actually known. One of them, however, is a direct alteration of the nutrition of the nerves. It is possible that the neuritic atrophy at the inflamed spots is also due to this; and the same is perhaps true of the degenerative atrophy which occurs in the optic, oculo-motorius, and other cerebral nerves in tabes, and which has perhaps the same origin as the gray degeneration of the posterior columns of the spinal cord, but which may also be due to a primary progressive atrophy.

Leber says plainly that optic nerve atrophy is not the simple result of compression of the optic nerve fibres from inflammatory exudation. The "spontaneously progressive nervous atrophy," to an interesting case of which Jaccoud devotes two long lectures, seems, from the results of the post-mortem examination, to have been produced not spontaneously but by compression of the spinal roots.

There is little to be said in regard to the *symptoms* of atrophy. Of spontaneous idiopathic atrophy of the peripheral nerves (with the exception of the optic), we are almost entirely ignorant, and its symptoms are equally unknown. The statements made by Jaccoud on this point were contradicted by the autopsy. Whether the general debility in cachectic conditions depends upon an atrophy of the peripheral nerves is at least doubtful; it is more probably of central origin, or is, perhaps, due to atrophy of the muscles.

The secondary atrophy, on the other hand, is only a subsidiary symptom of the primary disease, and belongs to its general picture. The primary symptoms of irritation, and secondary symptoms of paralysis, which by many authors are ascribed to the atrophy, and accord so well with the general scheme, are consequently essentially symptoms of the primary disease. With this we are not here concerned, but must refer to the earlier sections of this work. (See especially that on

"Paralysis.") This much may, however, be stated with certainty, that, wherever atrophy is developed, the function of the nerve is lowered or altogether lost, and this condition is expressed by the terms paralysis, anæsthesia, amblyopia, and amaurosis.

It is not uninteresting to note that the atrophy, in the motor nerves at least, can be demonstrated by electricity, for when the reaction of these nerves to faradic or to galvanic excitation is considerably below the normal, or is altogether lost (presupposing that the muscles are not completely atrophied), we can, according to our present knowledge, fairly conclude that a condition of degeneration and atrophy of the nerves exists. Attention has been repeatedly called to this in speaking of traumatic and rheumatic paralyses, of paralysis from compression, of atrophic spinal paralysis, etc. This, if we disregard the results of ophthalmoscopic examination in optic nerve atrophy, is the only means we possess of directly diagnosticating atrophy of peripheral nerves. The proof of the presence of those diseases in which nerve atrophy may be anticipated with some degree of certainty, furnishes, of course, important additional evidence of its existence.

The *prognosis* of nerve atrophy varies with the nature of the primary disease. If the re-establishment of the connection of the nerves with the central nervous system, or recovery from the conditions causing the atrophy be possible, regeneration and restitution of the nerve itself may be confidently anticipated; but if these be impossible, or if the atrophy persist for a very long period, the prognosis is unfavorable. Many researches and experiments have shown that the tendency to regeneration of atrophic nerves is very great. Philipeaux and Vulpian have even observed partial regeneration in a portion of excised nerve implanted beneath the skin.

From all this it is obvious that the *treatment* of atrophy is essentially the same as the treatment of the primary disease. This must be treated, and if possible removed, and when we have accomplished this, the recovery from the atrophy may reasonably be expected. Little can be done in the way of treatment for the atrophy itself. Electricity will probably be found to be the most serviceable agent; but even this is only successful in secondary atrophy when there is a possibility of regeneration in consequence of the removal of the primary disease. It then acts by improving the conditions of the circulation, by exciting the functional activity of the nerves, and by molecular action upon them.

The galvanic current is pre-eminently active in producing these results. (See the general treatment of paralysis.) Certain electrotherapeutic experiments on optic nerve atrophy[1] appear to show that decidedly curative effects result, in many cases of (primary ?) atrophy of the optic nerves, from the use of the galvanic current.—Further, in the paralyses which so often accompany atrophy, energetic shampooing of the limbs, warm baths, saline baths, pine-needle baths, alternate warm and cold douches, friction, either alone or with spirituous and strongly irritating liniments, etc., may be recommended. All these can only be regarded as subordinate to the electric treatment, though no doubt they have often an action in relation to the causal indications of treatment, which should not be underestimated.

4. *Hypertrophy and Neoplastic Formations in the Nerves. True and False Neuromata.*

Rokitansky, l. c. p. II., pp. 493 u. 499.—*Förster*, l. c. II. pp. 640 u. 644.—*Hasse*, l. c. pp. 749 u. 756.

HYPERTROPHY: *A. Knoblauch*, de Neuromate et gangl. accessor. veris, etc. Diss. Francof. 1843.—*Hesselbach*, Beschr. d. pathol. Präpar. in Würzburg. Giessen, 1824.—*Moxon*, Guy's hosp. Rep. Ser. III. Band VIII.—*Ferréol-Reuillet*, s. Virchow-Hirsch Jahresber. f. 1869, Bd. II. p. 349.

NEUROMATA: *Virchow*, krankhafte Geschwülste. Bd. III. p. 233 et seq. 1867. (Contains full bibliographical references.)—*R. Maier*, Lehrb, der allgem. pathol. Anat. Leipzig, 1871, pp. 224 u. 379.—*O. Weber*, Pitha-Billroth, Handb. d. Chir. II. 2. Abth. p. 226, 1865.—*Odier*, Man. de méd. prat. Genève, 1803.—*Schiffner*, Oesterr. med. Jahrb., 1818.—*Barkow*, Nova acta phy-med. T. XIV. 1829.—*Aronssohn*, observat. sur les tumeurs envelopp. dans. les nerfs. Strasb. 1822.—*Knoblauch*, l. c. 1843.—*Moleschott*, Arch. f. phys. Heilk. Bd. VIII. 1849.—*Rob. Smith*, a treatise on the pathol., diagnosis, etc. of neuroma. Dublin, 1849.—*Houel*, *Lebert*, Gaz. des hôp. 1852, No. 17.—Mém. de la Soc. de Chir. T. III. 1853.

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[1] See the references on Electro-therapeutics in the recent annual volumes of Virchow-Hirsch, Jahresbericht über die gesammt. Medicin.

Wedl, Grundz. d. path. Histol. Wien, 1853, p. 726.—*Kupferberg*, z. path. Anat. d. Geschwülste im. Verl. d. Nerven. Mainz, 1854.—*Volkmann*, Ulcerirend. Neurom am Handteller. Virch. Arch. XII. 1857.—*Schuh*, Zeitschrift der Wiener Aerzte, XIII. 1857.—*Temoin*, *Wilks*, multiple Neurome s. Canst. Jahresber. pro 1858, III. p. 18.—*Dehler*, u. *Förster*, Würzb. med. Zeitschr. II. 1861.—*Hitchcock*, Americ. Journ. of med. Sci. 1862.—*O. Heusinger*. multipl. Neur., Virch. Arch. 27. 1863.—*O. Weber*, über Nervengeschw. Verh. d. naturh. med. Ver. Heidelberg, IV. p. 99. 1867.—*A. Heller*, mult. Neur. Virch. Arch. 44.

Hypertrophy of the peripheral nerves is of extreme rarity, and belongs rather to the category of anatomico-pathological curiosities than to practice. In such cases the larger and more voluminous nerves either exhibit true hyperplasia of the nerve-tissue, that is to say, increase in the number of the nerve-fibres, division or splitting up of the fibres into a large number of daughter fibres;[1] great thickness of the medullary sheath, and even of the axis cylinder; or, and this is perhaps the most common event, an increase of volume occasioned by interstitial hypertrophy of connective tissue. Both conditions, notwithstanding their great general pathological interest, are, up to the present time, wholly destitute of clinical interest.

Amongst the researches that have hitherto been made, it may here be mentioned that, in many instances of elephantiasis, remarkable thickening and hypertrophy of nerves, partly with transition-forms into neuromata, have been observed; that in multiple neuromata, whether hereditary or arising from other causes, diffuse hypertrophy has been found in the affected nerve, as in a case reported by Bischoff, and in one by Hesselbach,[2] in another also by Heller;[3] that Günsberg observed both sciatic nerves to be thickened and hypertrophic in a phthisical patient, whilst Laumonier observed the same condition in many of the nerves of a young man, and Moxon[4] in many of the nerves of a female subject; and, finally, that in the case already mentioned, reported by Ferréol-Reuillet, a remarkable hyperplasia of many nerves was observed.

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[1] A. Heller, Neumann and others.

[2] Cited by Virchow, Geschwülste, Bd. III. p. 261.

[3] Loc. cit.

[4] Virchow, loc. cit.

None of these cases possess any clinical value; no symptoms can at present be depended upon to indicate the existence of nerve-hypertrophy. Nothing, therefore, can be said in regard to treatment.

Neoplastic formations in the nerves are of much greater practical inportance than these hypertrophies. These, since the time of Odier, have been very generally termed *neuromata*, without regard to the kind of tissue of which they are composed. In accordance with the principles of the nomenclature of tumors, this term must be preserved for those tumors of nerves which consist exclusively, or, at least, chiefly, of true nerve-substance. Custom, however, and the practical identity of the various forms of tumors, have led to the adoption of the term "false neuromata," to distinguish those which are not composed of nerve tissue (but which are seated on the nerves and proceed from them) from the above-mentioned proper or true neuromata.

Both groups are of not unfrequent occurrence, but it is only in a certain proportion of cases that these affections acquire any considerable practical importance. They often exist without causing any symptoms; and since they are chiefly interesting in their anatomico-pathological and surgical aspects, we shall here, in accordance with the general plan of this work, discuss them with great brevity.

Pathological Anatomy.—Since the common term neuroma, introduced by Odier, has been applied to all neoplastic formations in the nerves, a complete sifting and critical arrangement of the material that has accumulated, such as has been attempted in the classical work of Virchow, is beset with considerable difficulties. It is necessary, in the first place, to separate, by a very sharp line of demarcation, the tumors that chiefly, or exclusively, *consist of true nerve-tissue* (neuroma verum), from those that are *composed of any other tissue* (neuroma spurium). The last expression might, perhaps, advantageously be allowed to drop into oblivion. Careful research shows that the following are the principal forms:

1. *True neuroma; neuroma verum.*—This consists chiefly of nerve fibres, intermingled more or less freely with connective

tissue, which is sometimes soft, sometimes tough, sometimes rich, and at other times poor, in vessels. From these differences various forms of neuroma result, such as fibro-neuroma, glio-neuroma, myxo-neuroma, neuroma-teleangiectodes, etc. It has not been quite certainly ascertained whether ganglion-cells occur in these neuromata. The origin of the newly-formed nerve fibres is partly referable to granulation-tissue, and partly to the increase and division of the already existing nerve fibres (Heller). True neuromata occur almost exclusively in spinal nerves, rarely in sympathetic nerves, and still more rarely in any of the cerebral nerves. Their size is extremely various, from that of a millet seed to that of the fist.

Histological investigation tends to the differentiation of two forms of true neuromata. In one, medullated double-contoured fibres are common (neuroma myelinicum of Virchow). These give to the tumor a medullary white aspect, as may be seen in the neuromata that form after amputation. In the other form, extremely fine non-medullated fibres are present, which usually form a confused and felt-like mass, strongly resembling fibro-myoma of the uterus. The color of this form of neuroma is gray (neuroma amyelinicum). The histological recognition of this tumor is extremely difficult, and was first facilitated, and indeed rendered possible, by Virchow. It was formerly mistaken for fibroma, for fibro-sarcoma. A great part of the so-called multiple neuromata belongs to this category of true non-medullated neuromata.

2. *Other neoplastic formations in the nerves* (*neuromata spuria*).—The characteristic feature of these is that the principal portion of the tumor does not consist of nerve tissue, and that the number of the nerve fibres proceeding from the affected nerve into the tumor does not appear to be augmented. Various kinds of tumors belong to this category :—

a. *Fibromata.*—These are composed of more or less dense connective tissue with a few nerve fibres, and have accordingly a more or less dense consistence ; they often appear in the form of small hard knots, and the majority of those neoplastic formations in the nerves which are characterized as tubercula dolorosa consist chiefly, or exclusively, of fibrous tissue. It is probable that formerly most amyelinic neuromata were included under the fibromata; but, even deducting these, fibromata constitute the most frequent neoplastic formations in the nerves.

The term *tubercula dolorosa* has long been applied to certain small knot-like neoplastic formations seated in the peripheral nerves, and especially in the small sensory cutaneous branches of the extremities, and in the vicinity of the joints; they are characterized by extraordinarily increased excitability, occurring in paroxysms, and induced by contact, change of weather, etc. It does not appear justifiable to regard these as a special form of tumor, since, whilst there is a general agreement in the symptoms, the utmost variety occurs in the histological characters. Some have been found which consist of more or less dense connective tissue, others which contain numerous muscle cells, and others with cavernous tissue. Some exhibit a structure resembling that of the Pacinian corpuscles, others that of fibro-cartilage; and, in regard to others, there can be no doubt that they belong to the true neuromata, or, at least, have a close affinity to them. Transitional forms of all kinds—in regard to their symptoms also—between these and the ordinary forms of neuromata, are also to be met with, so that it is advantageous to drop this category of neoplastic formations, and keep strictly to the histological classification.[1]

b. *Myxomata.*—These are tumors which are composed of mucous tissue with its characteristic features, and are not unfrequently observed in the nerves. They consist of soft, lobulated, reddish, transparent, gelatinous neoplastic formations, in which the microscope reveals the presence of fully formed stellate intercommunicating cells, sometimes adipose tissue, &c. Cystic formations are also of frequent occurrence in myxomata, and have led to the formation of a group termed neuroma cysticum. *Gliomata* have hitherto been observed with certainty only in the auditory nerve.

c. *Sarcomata* occur, in various forms, as cellular, fibrous, and similar forms of sarcomata in the nerves. They not unfrequently exhibit transitional forms between fibroma and myxoma, to the latter of which they present great similarity in their external aspects, and in their mode of development. Not unfrequently, blood-vessels are abundantly developed in their interior. Upon the whole, they are of rare occurrence.

d. *Carcinomata*, on the other hand, are more common and also present various forms. The cancroid is rare, scirrhus and medullary cancer more frequent, and carcinoma melanodes is also common. These are all forms of neoplasm that generally affect the nerves, secondarily penetrating them from adjoining parts, and spreading in the neurilemma, beneath which they form knot-like protuberances at various points. Nevertheless, primary independent carcinoma also occurs in the nerves. It is almost always accompanied by more or less complete destruction and degeneration of the nerve fibres.

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[1] See Virchow, loc. cit.

e. *Syphilitic gummata* not unfrequently form in the cerebral nerves at the base of the cranium, and are here for the most part propagated from the membrane of the brain, though they may also be developed in the nerves as more or less independent tumors.

f. *Lepra nervorum* seldom appears in the form of a separate tumor, but rather as a diffuse or more or less fusiform swelling of the nerves. It consists of a development of granulation tissue which is often indistinguishable from that produced in inflammation. (Perineuritis leprosa.)

In regard to the *size* of nerve tumors, it is obvious from the foregoing enumeration of their varieties, that it may be extremely various. In point of fact, they occur of all conceivable sizes, between that of a millet or mustard seed and that of a child's cr even a man's head. In the greater number of cases, however, nerve tumors vary in size from that of a bean to that of a fowl's egg, and hence are chiefly of small size.

Again, in regard to the *number* of tumors that may be present at the same time, the greatest variations exist. In very many instances, there is only one, a solitary neoplasm on some particular nerve trunk. This holds good, not only for the extremely painful small neuromata which answer to the name of tubercula dolorosa, but for all the varieties of tumors in nerves.

Nevertheless, it is more common to find many of these tumors present together, and sometimes they exist in large numbers, and they may be either *locally* or *generally numerous*. When *locally numerous* they may either form a series of knots in one and the same nerve, or what is more common, numerous knots in the various branches of one trunk or plexus. A subordinate form of this local multiplicity is represented by the so-called neuroma plexiforme, in which a great plexus of thickened and hypertrophied nerves are reunited into one plexiform mass. On the other hand they may be *generally numerous*, for they occur often in very large numbers, amounting sometimes even to several thousand, in all parts of the body, chiefly in the spinal, but occasionally also in the cerebral and sympathetic nerves. Such multiple neuromata have been especially found in the cauda equina, in the pelvic plexus, and the intermediate nerves have then for the most part been found to be thickened and hypertrophic. In this case the multiplication of the tumors is not to be regarded as an instance of malignity, as in malign tumors, but simply as a morbid disposition to neoplastic formation in a particular tissue.

The relations of these various tumors to the nerves may be very diverse, and are not always easily demonstrable. The new formation is either on one side of the nerve, so that the nerve seems to run in close proximity with it, or it occupies the centre of the nerve, the several fasciculi coursing over the surface of the tumor, or the nerve runs without any line of demarcation directly into the tumor, the fibres breaking up into a kind of brush or pencil. In true neuromata, either the whole or part only of the fibres of the nerve participate in the new formation; false neuromata proceed, for the most part, from the neurilemma, and the nerves may either remain more or less completely intact, or they may be compressed and completely destroyed. These various conditions will of course exercise a very marked influence on the symptoms.

The *etiology* of neuromata is still in many points obscure. In the first place experience teaches that a certain *predisposition* to them exists in many persons, especially in those who belong to families that are predisposed to neuroses. Their origin in congenital and hereditary states is not unfrequently observed, and certain relations to idiocy and cretinism must not be overlooked. Phthisical and scrofulous (though not tubercular) subjects appear to be especially predisposed to the formation of neuromata. A special tendency, or at least a local tendency of the tissue to multiple neuromata must be admitted, though it is unknown upon what this depends. Isolated neuromata are more common in women, whilst multiple neuromata occur almost exclusively in men. Neuromata occur at all ages, and are often congenital.

Of the *direct causes* the best known are traumatisms, such as blows, pressure, bruises and the penetration and retention of foreign bodies in the nerves, as fragments of glass, points of needles, ligature threads, etc. These have all been observed to constitute causes of neuromata with sufficient frequency to render their action certain. To this category belong also the cicatricial neuromata which form at the cut ends of divided or otherwise injured ·nerves, and the common neuromata, which appear in the form of rounded or elongated swellings of the divided nerve ends in the cicatrices of the stumps in amputation. They may be regarded to some extent as the expression of the intensity and energy of the regenerative processes which are taking place in the divided nerve ends.

These neuromata are in part also to be regarded as inflammatory products. A chronic neuritis may thus occasionally constitute the starting-point of a neuromatous growth. In accordance with this, neuromata have been observed in the neighborhood of parts undergoing chronic inflammation, as for example in caries of the wrist joint.

The origin of neuromata in rheumatic inflammation, which was formerly very generally admitted, is, at least, of doubtful occurrence, and is, perhaps, only intelligible as resulting from the intervention of a rheumatic neuritis. It has been already mentioned that syphilis, lepra, and elephantiasis occasionally lead to the formation of tumors in the nerves. Even after all these causes have been mentioned, it still remains a fact that in many, perhaps even in the majority of cases, no definite cause is demonstrable, and we are compelled to admit a spontaneous origin of the new growth.

The symptoms of tumors in the nerves are very variable. Many cases present no symptoms at all throughout their whole course, whilst others are the cause of persistent and severe suffering to the patient, without any intelligible reason. Such differences are, however, it is probable, immediately due to variations in the finer relations between the nerves and the tumor.

The history of isolated neuromata, if known at all, is usually that of severe and incurable neuralgia; hence the term applied to them of tubercula dolorosa, which, as we have already seen, is only adapted for such cases as present these symptoms.

In cases of this kind the principal symptom consists of violent pain, gradually increasing in intensity, and radiating from certain points towards the periphery. The pain may present various characters, being tearing or lancinating, or a deep-seated aching, with boring or burning sensations. It is almost always either remitting or completely intermitting. It is increased by cold and damp weather, by pressure, by the manipulation requisite for the investigation of its nature, and in women, in many instances, by the return of the menses, or by pregnancy. It may often be made to disappear temporarily by firm pressure on the nerve above the tumor. Its intensity varies considerably in different instances, being often most severe in very small tumors on peripheral cutaneous branches. Variations in its intensity, which cannot here be fully described, often occur in the course of the disease.

In addition to the pain, *parœsthesiœ* are experienced, though, upon the whole, not very frequently; still, in many cases a feeling of numbness, formication, and sensations of heat and cold are perceived in the area of peripheral distribution of the affected nerve. *Motor disturbances* are of still less frequent occurrence, and while absent in many cases, and, indeed, in all cases of neuromata of purely sensory nerves, they appear as reflex phenomena of motor irritation in the form of tremors, spasms, contractures, etc., which ultimately pass into various grades of paralysis. Paralytic symptoms also appear in most sensory regions—a more or less widely extended anæsthesia completing the general picture of the disease. This not unfrequently occurs in the form of anæsthesia dolorosa, especially in malignant disease.

It is only in extremely severe cases, or in very excitable persons, that widely radiated symptoms occur as diffused pain in the head and spine, and in the seats of distribution of very remote nerves. Neither vertigo, nor epileptic attacks, nor hysterical convulsions are of common occurrence during the paroxysms of pain, and tetanic conditions are still less frequent.

Direct examination in such cases not unfrequently reveals the presence of a small, movable, usually very painful, knot, in the nerve, which is extremely tender to the slightest touch, and from which paroxysms of pain may proceed. In other cases, a neoplastic formation of larger size may be readily recognized. If the neuroma, however, happen to be seated in some part of the nerve which is inaccessible to palpation, as, for example, in the pelvis, in the cauda equina, etc., its existence cannot be objectively demonstrated.

The further course of the disease varies in different instances: the symptoms may gradually rise in intensity, so that the patient becomes a martyr to the most frightful suffering, whilst the persistent want of sleep, secondary disturbances, etc., lead gradually to a high degree of cachexia, and, by progressive exhaustion, even to death.

Or, the symptoms may gradually remit, and ultimately cease, and the patient may be said almost to be reanimated. In a few cases, the tumor entirely disappears.

Or, symptoms of paralysis gradually supervene, which are more or less extended, according to the seat of the tumor, as, especially, in neuromata in the cauda equina. In such cases, paraplegia may be observed, with muscular atrophy and every variety of trophic disturbance, with inability to stand, and with paralysis of the bladder and rectum, which gradually lead to a fatal termination.

True neuromata are not infectious, and exert no deleterious influence upon the organism, in cases where they cause no disturbance by their position, or in a mechanical manner; their duration may be unlimited, and they may remain stationary for many years without, in any way, injuring the general health. Many cases, however, exhibit a tendency to relapses, and then often appear in large numbers, either in the same or in adjoining nerves. In these cases the skin may become adherent, and ulceration of the neoplastic formation may occur.

In the heteroplastic new formations, the character of the neoplasm in the nerve naturally determines its course, and they may have a deleterious influence without occasioning any remarkable nervous disturbance. In many instances, however, violent symptoms, as acute neuritis, etc., occur, if the larger nerve trunks become implicated in carcinoma or similar malignant disease. The disease spreads rapidly to the central nervous system, and then a sudden increase of the cachexia is the not uncommon result.

Multiple neuromata present, at least in many cases, scarcely any symptoms. They are often only accidentally discovered in autopsies, or during life. These tumors may, however, cause many disturbances by their mechanical action, evidenced by pain, paralysis, anæsthesia, atrophy of the muscles, cutaneous ulcerations, etc., all of which have been observed. In other cases, and especially when the sympathetic nerves are seriously implicated, general debility and anæmia, disturbances of the digestion, etc., occur, and the patient succumbs rapidly, with symptoms that are not easily stated. It is possible that the implication of the vagus, sympathetic, phrenic, etc., exerts an influence on this course. Multiple neuromata may last many years, and even decades, without causing great disturbances, whilst, on the other hand, they sometimes speedily lead to a fatal termination.

The *diagnosis of neuromata* is founded, in addition to the above-named symptoms, almost solely upon the presence, on certain nerves, of round or oval tumors of various sizes, which are movable from side to side, but not in the direction of the length of the nerve. An exact anatomical diagnosis of the neoplasm is seldom practicable, and must be based on the general principles of the diagnosis of tumors; it can, however, be accomplished with certainty by means of the microscope. When no tumors can be demonstrated by external examination, the presence of a neuroma can only be concluded with some probability from the existing nervous disturbance.

Prognosis.—True neuroma is always a local and benign affection. It may, however, return after extirpation once or several times, and finally the skin may become adherent to it and even ulcerate. No instance is known of its producing general infection.

The prognosis of other neoplastic formations in nerves is the same as when they develop elsewhere, and here also the greater or less degree of heteroplasia of the tumor is of the greatest importance in founding the prognosis; specific malignant neoplasms have always a bad prognosis.

The prognosis is bad also when the neuralgic pains are severe, and when extirpation is impracticable, because, in this event, it is impossible to subdue the pains, and the strength of the patient is gradually worn out. The prognosis otherwise depends on the intensity and extent of the nervous and trophic disturbances. When no operation can be undertaken, the patient's life is made intolerable. This does not, of course, apply to the not unfrequent cases where the neuroma produces no symptoms.

Treatment.—Experience has shown that all attempts to disperse or reduce the size of these tumors, or to cause them to disappear, either by internal remedies or by external applications, are futile; and it is therefore unnecessary to spend much time on such means. Surgical treatment constitutes the only rational method of dealing with neuromata, namely, the extirpation or destruction of the tumor. The only objections are, that surgical means are not applicable in all cases, and that they do not protect from relapses and extension of the disease. The most favorable results are of course obtained in simple or quite local multiple tumors that are accessible to operation.

Extirpation is by far the best method of treatment, and it succeeds best where the tumor can be removed and the nerve left intact. If this be impracticable, the smallest possible portion of the nerve should be extirpated; it is worthy of notice, however, that even when a portion of nerve measuring an inch in length has been removed, regeneration has taken place, though, perhaps, only after the lapse of a considerable period. Frequent relapses compel the performance of repeated extirpation, and even of amputation. The neuromata that occur after amputation should be extirpated; it is seldom requisite to amputate at a higher point. In multiple neuromata, or when the tumors are very deep-seated, an operation will be undertaken with caution, or the most painful ones will alone be removed. For all details, reference must be made to text-books on surgery.

Destruction of neuromata by caustics or by electrolysis is, under all the circumstances, not very advisable, unless indeed extirpation by the knife is impossible.

Apart from these measures, symptoms alone can be treated, and a careful physician may materially alleviate the sufferings of the patient, which are often truly terrible. On this point, we may refer to what has been said respecting the treatment of neuralgia, of anæsthesia, and of paralysis. In such cases, we must chiefly rely on the varied and cautious use of narcotics, and it is unnecessary to repeat here the general rules in regard to their use.

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